

WV
5
A512p
1922

WV 5 A512p 1922

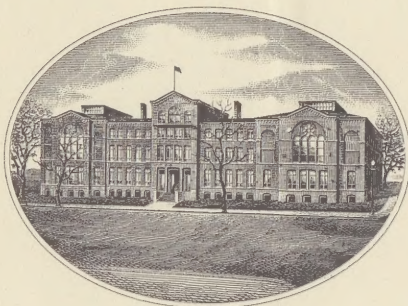
51310760R



NLM 05267745 0

NATIONAL LIBRARY OF MEDICINE

NATIONAL LIBRARY OF MEDICINE
Washington



Founded 1836

U. S. Department of Health, Education, and Welfare
Public Health Service

PRE-SESSIONAL PAPERS

AMERICAN OTOLOGICAL SOCIETY

WASHINGTON, D. C.

May 1st, 2d and 3d

1922

DR. GEORGE E. SHAMBAUGH

DR. ISAAC H. JONES

DR. L. W. DEAN

DR. J. GORDON WILSON

PUBLISHED BY THE SOCIETY

GEO. H. REYNOLDS

PRINTING

NEW BEDFORD, MASS.

1922



44
PRE-SESSIONAL PAPERS

to sale
96
AMERICAN OTOLOGICAL SOCIETY

WASHINGTON, D. C.

May 1st, 2d and 3d

1922

DR. GEORGE E. SHAMBAUGH

DR. ISAAC H. JONES

DR. L. W. DEAN

DR. J. GORDON WILSON

PUBLISHED BY THE SOCIETY

GEO. H. REYNOLDS

PRINTING

NEW BEDFORD, MASS.

1922

WV

5

A512p

1922

c.1

NATIONAL LIBRARY OF MEDICINE
WASHINGTON, D. C.

REVIEW OF OUR KNOWLEDGE OF THE BLOOD VESSELS SUPPLYING THE INTERNAL EAR.

By GEO. E. SHAMBAUGH, M. D., Chicago, Ill.

I have been asked to prepare a statement of our present knowledge concerning the blood supply of the labyrinth. The subject is largely one of purely scientific interest but is not without its practical bearing on clinical otology. My own interest in the blood vessels of the labyrinth was aroused in the first place while studying cases of the apoplectiform type of internal ear disease ordinarily designated as Meniere's disease and where the presumption formerly was that the disturbances were the result of hemorrhages into the labyrinth. An increasing knowledge of the labyrinth and especially of the symptomatology of sudden disturbances of the internal ear, especially those in connection with invasion of the labyrinth by infection, such as the extension from suppurative otitis media, has established on a firm basis the proposition first advanced by Meniere, that a hemorrhage into the labyrinth is capable of producing the typical Meniere-symptom-complex. This symptom-complex consists of the sudden onset of deafness associated with tinnitus and vertigo. It has become equally clear on the other hand that this symptom-complex is produced by any sudden involvement of the internal ear, such as might result from traumatism, or from an extension of infection from some neighboring part as well as the toxic action of infection on the eighth nerve or the action of certain drugs.

Before entering into a discussion of the details of the labyrinthine blood vessels I desire to call attention to certain facts in connection with this blood supply which should be kept in mind when considering the possible clinical effects of alterations in this supply.

The first point of interest in this connection is that the

entire labyrinth is supplied by a single vessel which enters the internal ear by way of the meatus acusticus internus. It is evident therefore that a tumor developing in the internal meatus might disturb the function of the labyrinth not alone by exerting pressure on the eighth nerve but by interfering with the blood supply of the several end organs of the internal ear. An embolus lodging in this artery would result in a sudden suppression of function and give rise to the Meniere-symptom-complex in its most characteristic form. The deafness would be complete and permanent and the vertigo intense, though of course the latter would be of but temporary duration.

A second anatomical fact of interest because of its possible clinical bearing is that the several divisions of the labyrinthine artery going to supply different parts of the labyrinth are in the nature of end-arteries, that is there exists no anastomosis with other vessels except through the terminal capillaries. This is the type of arterial supply to the brain and certain other organs. This anatomical fact makes it quite clear that an embolus, such for example as the air-embolus in cussion disease, when lodging in a particular branch of the labyrinthine artery will produce an infarct in the region supplied by this particular artery and cause a suppression of function of the end organs restricted to the region which this vessel supplies. For example, a single arterial trunk called the anterior vestibular artery is the sole blood supply for the macula acustica of the utricle and the crista acustica of the horizontal and superior canals. An embolus lodging in this artery will produce disturbances of equilibrium and vertigo, but will not affect the function of the cochlea. Every carefully observing otologist with any extensive experience has encountered just such cases clinically, which apparently might be caused by such a lesion as the one just described, or possibly by some selective toxic action affecting the vestibular nerve but not the cochlea branch of the eighth.

The arterial supply of the cochlea consists of a large number of stems which, leaving the main vessel in the

modiolus, radiate at regular intervals out along the lamina spiralis ossea to supply the organ of Corti which rests on the membranous extension of the osseus plate, known as the membrana basilaris. Each one of these radiating stems are actually end arteries an obliteration of which will bring disturbance to but a restricted area of Corti's organ. From our knowledge of the physiology of the cochlea we would conclude that such a lesion will produce a defect more or less restricted in the midst of the tone scale. Here again is a clinical phenomenon with which otologists are quite familiar. I have had opportunity to study several cases where individuals apparently in normal health and with no history of any previous ear trouble developed a sudden involvement of one ear causing tinnitus aurium with a depression of the hearing function and where the functional examination demonstrated a lesion evidently in the labyrinth causing a defect restricted to the lower end of the tone scale. The location of the lesion in the cochlea itself seemed all the more certain because in most of these cases the symptom of diplacusis was also observed. Such a clinical complex might be accounted for on the assumption of an embolus in, or a hemorrhage from the artery supplying the apex of the cochlea.

Another point in connection with the blood supply of the labyrinth, this is branches from the labyrinthine artery and on clinical otology has to do with the question of anastomosis between the vessels supplying the endosteum of the labyrinth, this is branches from the labyrinthine artery and those found in the osseous wall surrounding the internal ear. It is apparent that where such anastomosis exists we would have a plausible explanation for extension of infection from the middle ear to the labyrinth without the formation of a bony fistula or of a rupture through one of the labyrinthine windows for the reason that blood vessels supplying the mucous membrane of the middle ear cavities also supply the underlying bone. Politzer and others have believed that such communications do exist between the tympanum and the inner ear. Some years ago I devoted

a special research to this problem and was able to demonstrate a great many places where the labyrinthine artery sent branches into the surrounding bone. This condition was found however only in the calf. In neither the pig nor the sheep, both of which I was studying extensively at that time was there any evidence that the artery for the labyrinth gives off branches to the surrounding bone. Since that time I have worked out the blood supply for the labyrinth of the dog as well as the human and in neither of these have I been able to demonstrate any vessels from the interior of the labyrinth which supplied the bony capsule. This fact if true, closes the door for any possible communication between the blood supply of the internal ear and that of the mucous membrane lining the middle ear.

Another fact in connection with the blood supply of the labyrinth deserves special attention because of its fundamental bearing on the problem of sound perception. This is the fact that the membrana basilaris is always a vascular structure. It is true that the so called zona pectinata, that is the part of the basilar membrane lying external to the tunnel of Corti has in some of the animals thus far studied been found free from blood vessels. I found vessels in the zona pectinata in the pig, the sheep and the calf but I did not observe these either in the dog or man. Nabeya found the zona pectinata vascular in the rabbit but not in the guinea pig. The important point is that in all the animals studied, including man, the part of the basilar membrane directly under Corti's organ, that is under the tunnel of Corti, always contains blood vessels. The reason why the vascularization of the basilar membrane is of special interest is because this is the structure fixed upon by Helmholtz and his followers as the vibrating mechanism which by responding according to the principle of physical resonance in its several parts to tones of varying pitch, brings about the stimulation of different areas of Corti's organ for each tone of the scale. It is of course essential that this vibrating mechanism should always respond the same to the same impulse, otherwise there could be no

recognition of pitch. It requires no argument to prove that a vascular structure such as the basilar membrane is incapable of reacting the same at all times to the same impulse. So that whatever our conclusions may be in speculating on the function of sound perception, one thing is quite definite, namely that the hypothesis that the basilar membrane in the vibrating mechanism is rendered untenable because of the fact that this membrane is a vascular structure.

I desire to call attention to but one more fact in connection with the blood supply of the labyrinth which has an important bearing to clinical otology. We have in the membranous labyrinth, a highly vascular structure suspended in a fluid which completely fills these osseous chambers. In the bony walls of the labyrinth there exist several openings suitable for relieving an increase in the intra-labyrinthine pressure. These openings are the oval and round windows, the aqueductus cochlea and the aqueductus vestibuli. Through the latter passes the ductus endolymphaticus, a tubular prolongation from the membranous labyrinth which on the posterior aspect of the petrous bone opens into a veritable expansion tank apparently designed especially for taking care of fluctuations in the intra-labyrinthine pressure. From this description of the relations of the internal ear, it is apparent that with each pulsation of the heart there must be produced in the labyrinth a to-and-fro movement of the endolymph. The bearing which this fact has to clinical otology is as follows. One of the most important functions of the vestibular mechanism is to supply a constant stream of tonus impulses to the skeletal muscles so necessary in preserving the normal equilibrium. It seems probable that this tonus function of the semicircular canals is a much more vital and important function than that of giving information regarding turning movements and position in space. That these tonus impulses emanate from the crista acustica is generally accepted but what produces the constant stimulation of the hair cells of the crista has never been satis-

factorily explained. Ewald ventured the suggestion that these cells were kept in a constant state of stimulation as the result of endolymph currents which he assumed were constantly present as the result of the constant replenishing of the endolymph from the stria vascularis and its escape through the ductus endolymphaticus. It is only necessary to point out that such a current could not exist for the simple reason that the ductus endolymphaticus ends in a closed sack and does not communicate with the cerebrospinal fluid as Ewald assumed. Barany accepts the conclusion that a constant stream of tonus impulses emanate from the hair cells of the cristae but offers no explanation for the continuous stimulation of these hair cells which this hypothesis presupposes. The explanation given above of a continuous to-and-fro movement in the endolymph resulting from the arterial pulsations supplies exactly the sort of stimulation necessary to account for the constant supply of tonus impulses which as we have already pointed out constitutes such an important part of the vestibular function. In my work on the structure and function of the crista ampullaris (1) I offered this explanation of the origin of labyrinth tonus and at the same time showed how the sum total of tonus impulses from the labyrinth on one side stimulates the muscles causing movements toward the opposite side thus accounting for the clinical phenomena observed in the sudden destruction of one labyrinth when the patient falls toward the affected side and a spontaneous nystagmus develops with the slow component towards the affected side. I repeat the explanation here as it seems to be a point not fully appreciated by many otologists.

The facts may be set forth briefly as follows: My studies of the minute anatomy of the crista ampullaris convinced me that an actual displacement of the cupola on the crista as generally supposed, where-by all the hair cells on each side of the crista are stimulated is not possible. This hy-

(1) *Über den Bau und die Funktion de Crista Ampullaris. Zeitschrift für Ohrenheilkunde, etc., Mai, 1912.*

pothesis of displacement of the cupola had given rise to the theory of a positive and negative response for the hair cells of the crista depending on the direction of the endolymph current. This is not sound physiological reasoning to assume that the same hair cells are capable of one kind of response by an endolymph current in one direction of an opposite kind of response by a current in the opposite direction. With my conclusion that a displacement of the cupola is impossible it is apparent that only those hair cells on the side of the crista receiving the impact are stimulated by an endolymph current while the hair cells on the opposite side of this crista are stimulated by an endolymph current in the opposite direction. With this fact in mind the explanation why the sum total of the tonus impulses from the right labyrinth produce movements towards the left and vice versa becomes quite clear. The explanation is as follows: It has been established that an endolymph current in one direction in a semicircular canal stimulates the muscles which produce movements in the direction of the endolymph current while a current in the opposite direction stimulates the opposite group of muscles. Furthermore it has been established that for each canal an endolymph current in one direction produces a much stronger response than does a current in the opposite direction. It happens now that for each canal it is those endolymph currents which stimulate muscles resulting in movements towards the opposite side that give the stronger responses which is only another way of saying that the impulses emanating from those hair cells of each crista, the stimulation of which produces movements toward the opposite side, are stronger than the impulses from the opposite group of hair cells which produce movements towards the same side. The tonus impulses from the crista of each canal stimulates muscles causing movements in the two directions but the stronger impulses are of course always those causing movements towards the opposite side. Barany in a recent communication (1) re-

(1) Modern Labyrinthology—Laryngoscope, July, 1921.

ports a series of observations bearing on the effect of the vascular supply of the labyrinth on labyrinth tonus.

On page 409 he states that "this (phenomena of ocular movements) is due to the to-and-fro movements of the endolymph and that this movement is brought about by the congestion or depletion of the vessels in the labyrinth. It may be assumed that the endolymph is directed towards the vent during systole and away from it during diastole. As the result of this observation I think the hypothesis of Shambaugh advanced some years ago may be realized, namely, that the pulse wave in the normal subject influences the endolymph in a to-and-fro motion and thereby establishes the tonus of the vestibular apparatus from the periphery. Apparently these movements are so slight, due to the small calibre of the normal vessels, that they do not produce a slow ocular movement in the normal subject, nevertheless I think it might be possible to maintain tonus of the vestibular apparatus from the periphery with even such a slight stimulation. In the presence of hyperemia of the labyrinth these movements naturally are more marked and then set up this rocking ocular movement. The presence of a peripheral labyrinth tonus (Shambaugh) does not indicate, as I would observe in passing, a coexistent central tonus of the nerve cells." With this brief statement of the bearing which the blood supply of the labyrinth has on clinical otology, we approach with added interest the study of these blood vessels.

THE ARTERIAL SUPPLY.

This comes from a single vessel, the labyrinthine artery which reaches the internal ear by way of the meatus acusticus internus. I have seen no exception to this arrangement except in the calf, where as an anatomical variation an arterial trunk was seen to penetrate the capsule of the labyrinth and form the artery of the crus commune.

As regards the distribution of the branches of the labyrinthine artery to the several parts of the labyrinth, a striking uniformity exists for all the animals so far studied. In

each species including man the first branch constitutes the sole blood supply for the macula acustica of the utricle as well as the cristae acusticae and the anterior crurae of the horizontal and superior semicircular canals. This vessel is known as the anterior vestibular artery.

The crista acustica of the posterior canal is supplied by the second branch known as the posterior vestibular artery. This vessel supplies not alone the crista acustica of the posterior canal but also the crus commune, the posterior crurae of the posterior and horizontal canals and both crurae of the posterior canal. There are a number of variations in this arterial supply for the crus commune and posterior canal. The most typical arrangement is for a single artery to supply all of these structures. In some animals as for example the sheep and the calf, the posterior vestibular artery supplies only the crista and the posterior crus of the posterior semicircular canal, while a separate branch from the labyrinthine artery supplies the crus commune and the posterior crurae of the superior and horizontal canals as well as the anterior crus of the posterior canal. The position of the artery supplying the semicircular canals is always the same. They lie in the endosteum along the concave side of the canal.

The arrangement of the arterial branches at the base of the cochlea differ widely in different species. In both the calf and the pig an elaborate system of anastomosing loops exists from which are given off all the arteries to the cochlea as well as to the vestibule and semicircular canals, with a single exception the anterior vestibular artery, the first branch of the artery of the labyrinth is given off before the vessel breaks up into anastomotic loops. In the sheep these anastomotic loops are very meagre as they also are in the dog and in man.

The typical arrangement for the arterial supply of the cochlea consists of two sets of radiating arteries. One set runs out along the lamina spiralis to supply the spiral ganglion and the organ of Corti, the terminal twigs forming one or two so-called spiral vessels attached to the under surface

of the basilar membrane directly under the tunnel of Corti. From these spiral vessels an occasional branch radiates across the zona pectinata of the basilar membrane to join the vessels in the spiral ligament. These vessels in the zona pectinata I found in the pig the sheep and the calf but not in the dog or in man. Nabeya found these in the rabbit but not in the guinea pig.

The situation as regards the vascularization of Reissner's membrane is peculiar. This structure remains as a rule free from blood vessels. In some species however, it is a vascular structure. In the human labyrinth no vessels are found in Reissner's membrane. This is also true for the domestic pig, the guinea pig and the dog. In the sheep, the calf and the rabbit, Reissner's membrane is vascular. The arterial supply comes either from the vessels radiating over the scala vestibuli or from branches from the stria vascularis while the veins join those of the lamina spiralis.

The under surface of the basal coil is usually quite free from arterial vessels. In the sheep and the calf, I found a few arterial twigs lying in the endosteum of the basal coil, external to the veins occupying this region. In both of these species an arterial twig is constantly found leaving the labyrinth along the aqueductus cochlea. It is not unusual also to find a small arterial twig leaving the labyrinth along the aqueductus vestibuli. I found such a vessel in both the sheep and the calf, but did not observe this in the pig, the dog or in man.

THE VENOUS SYSTEM.

The venous system of the labyrinth can be summed up in a very few words. In most of the species so far studied all the veins from the labyrinth are collected into a single vessel which leaves the internal ear by way of the aqueductus cochlea. This condition exists in the pig and the sheep as well as in the guinea pig, the dog and the cat. A variation from this rule is found in the rabbit and in man, where the chief exit of the veins from the semicircular canals is along the aqueductus vestibuli. In the calf a

single vein, that of the aqueductus cochlea is the rule, and as an anatomical variation, occasionally a small vein from the crus commune leaves by way of the aqueductus vestibuli. Siebenman for the human ear and Asai for the dog and the rat have described a third vein which drains the labyrinth lying in the meatus acousticus internus. Neither Nabeya nor myself have been able to verify this vessel. No vessel leaving the labyrinth was found by me in the internal meatus of the pig, the sheep and the calf. I have since made a series of preparations for man and the dog. In both I found all the veins leaving the labyrinth along the aqueductus cochlea or the aqueductus vestibuli. Nabeya found in both the guinea pig and the cat that all the veins of the labyrinth were collected into a single trunk, the vein of the aqueductus cochlea. In the rabbit only did he find a vessel draining the semicircular canals which found its way through the aqueductus vestibuli.

A summary of the more important features of the blood supply for the internal ear includes the following points:

(1). The arterial supply is from a single vessel entering the labyrinth by way of the meatus internus. An exception in the nature of an anatomical variation was found by me in the calf where an artery from the bony capsule supplied a branch to the crus commune. A similar observation was made by Sappy.

(2). The typical arrangement for the venous system is for all the vessels to collect into a single trunk which leaves the labyrinth along the aqueductus cochlea. An exception to this arrangement is found in some species including man, where the veins from the semicircular canals in part or as a whole are collected into a trunk which leaves along the aqueductus vestibuli.

(3). No communications exist between the blood supply of the labyrinth and the surrounding structures. Exceptions to this rule occur occasionally. In the calf many branches from the labyrinthine artery were given off to

the bony capsule. In the sheep, the calf, and the dog, a small arterial twig left the labyrinth along the aqueductus cochleae.

(4). The arterial supply to the several parts of the labyrinth is through end arteries. The several end-organs isolated in this way are: (a) the macula acustica of the vestibule, and the crista acustica of the superior and horizontal canals; (b) the crista acustica of the posterior canal; (c) the several parts of Corti's organ, each supplied by a vessel which radiates out from the modiolus along the lamina spiralis; (d) the macula acustica sacculae.

(5). The membrana basilaris is always a vascular structure, blood vessels being constantly found under the tunnel of Corti. The importance of this fact is that these vessels make it impossible for this structure to fill the role of the vibrating mechanism in sound perception, since to fill this role it is essential that it should vibrate the same at all times to the same impulse; and this a vascular structure would not be able to do.

CERTAIN NEURO-OTOLOGIC PROBLEMS.

By SAMUEL D. INGHAM, M. D. and ISAAC H. JONES, M. D.,
Los Angeles, California.

(To be read before American Otological Society, May 1922,
at Washington, D. C.)

OUTLINE

- I. Remarks about the new plan—the presentation of a broad subject, to which an entire session of the Society shall be given.
- II. The scope and limitations of this paper. This paper is designed to include all the main subjects included under Neuro-Otology. The limitations of the paper. It will make no attempt to be encyclopedic; its point of view is purely clinical; certain experiments in physics and upon animals are included, but only to the extent that they throw light upon clinical problems.
- III. Introductory remarks as to the part played by experimental research and clinico-pathologic research.
- IV. Problems—taken up one by one as entirely separate entities, but conforming to a general plan.
- V. Summary. An attempt to make the following classification, on the basis of the subject matter presented in this paper:
 - (a) What we are to regard as purely theoretic.
 - (b) What we are to regard as highly probable.
 - (c) What we can regard as reasonably proven—including the phenomena on which we feel we can rely in clinical diagnostic work.

I. REMARKS ABOUT THE NEW PLAN OF PRESENTATION.

The council of the Otological Society considered that an experiment might be tried at this meeting of the Society. Instead of having a series of papers on different subjects, the council considered that our program would be improved if we concentrated our attention upon a few main subjects, devoting one session, or a large part of a session, to the discussion of a single subject. Further, that as far as possible, one member should be selected for preparing a formal presentation of the subject. It was further suggested that this paper be printed in advance of the meeting and copies put in the hands of all the members of the Society, so that they would come to the meeting prepared to add as much as possible to the discussion. In this way, it is hoped that such a paper, followed by a large and more or less prepared discussion by many members, might constitute a fairly elaborate symposium of this subject.

Such presentation and discussion of neuro-otologic studies seems opportune. There is no other phase of otologic work more in need of such a discussion than the problems of neuro-otology. The problem involved in the study of the internal ear and its intracranial connections is still in its infancy. On the one hand, very little has been definitely established; on the other hand, when we come to learn more and more definitely the significance of the phenomena that we actually see in the examination of patients, we can look forward to an enlarged sphere of the usefulness of otologic study. To be sure, at this date, our conception of neuro-otology is not so vague as it was only a few years ago. To those who have made such studies, it is encouraging to note that a certain semblance of order has already taken the place of what was so recently a complete chaos.

II. THE SCOPE AND LIMITATIONS OF THIS PAPER

This paper is designed to present all the main subjects included under Neuro-Otology. Broadly speaking, neuro-otology includes every investigation that bears in any way on the internal ear itself; the anatomic connections of the internal ear with the intracranial structures—in other words, the complete

auditory apparatus as well as the complete vestibular apparatus; the normal physiologic functioning of the auditory apparatus and the vestibular apparatus; and the phenomena observed in pathologic change in any portion of either the auditory or vestibular apparatus. In studying the literature, it is surprising to note how little advance has been made in the knowledge of the intracranial portion of the auditory apparatus. It is equally surprising, on the contrary, to note that there has been a remarkable advance in the study of the vestibular apparatus; this is especially true during the past five years. At the present time, as much as ten per cent. of all otologic literature seems to be made up of vestibular studies, by leading investigators, of many nationalities, notably the Dutch, Austrian, Italian, Belgian, French, Scandinavian, German, Japanese, English and American. Instead of occupying a very casual position in otology, vestibular studies now include so many investigators and such a mass of work as almost to constitute a specialty of its own.

For this reason, this paper will make no attempt to be encyclopedic or to refer to the extensive bibliography. It will attempt to present only the essentials of each main neuro-otologic subject. Its point of view is the purely clinical; certain experiments in physics and upon animals are included, but they were undertaken and are presented only in the attempt to throw light upon clinical problems that come up in usual otologic practice.

III. INTRODUCTORY REMARKS AS TO THE PART PLAYED BY EXPERIMENTAL RESEARCH AND CLINICO-PATHOLOGIC .. RESEARCH. ..

The comprehensive neuro-otologic problem is to attain knowledge of the anatomy and physiology of the auditory and vestibular mechanisms. The immediate anatomic problem is, through what structures, along what various pathways and to what neural termini, do the many and varied impulses proceed from the internal ear. The object of such study is to enable us to interpret the significance of pathologic disturbance of the various portions of each mechanism.

There are two main avenues of approach to this problem—the experimental and the clinico-pathologic. These two methods are essentially similar. Not only have they the same object to attain, but the underlying principles are identical, in that they both include the study of structures in relation to function, and the study of structures in relation to the loss or disturbance of function. They differ in that the experimental is largely confined to animals. It might be well to consider the advantages and also the limitations of each of these two methods of approach.

EXPERIMENTAL RESEARCH.

Experimental research has certain advantages over the clinico-pathologic, presenting as it does the opportunity either to stimulate or to destroy certain tissues, and then to study the results. These results can be studied by noting changes or loss of function, and also by alteration or impairment of reactions to stimuli. In addition, it is then possible, by post-mortem examination, to make gross and histologic study of the structures affected. The further advantage of such experimental research is the opportunity afforded to establish a series of observations and to obtain a general average of results from many similar experiments.

Much important knowledge of the vestibular apparatus has already been attained by the experimental method. This includes observation and classification of the responses which can be obtained from a direct stimulation or destruction of separate structures within the internal ear. S. S. Maxwell has made recent valuable contributions to this phase of the subject, by experimental work on fishes; he will present a resumé of his work and that of others, in the discussion of this paper.

Another result of experimental work has been the tracing of degenerated nerve-fibers from the VIII Nerve and its connections, in various directions within the brain-stem and the cerebellum. The most recent and valuable work on this subject is that of Sachs and Alvis; Dr. Sachs will open the discussion by presenting the present status of available information from experimental work on animals.

There are many difficulties peculiar to vestibular study, that are met by one undertaking experiments upon animals. There is the difficulty of applying the principles of comparative anatomy and physiology; due consideration must be given to the variations of structure and function in the different animal species, including man. For example, the range and complexity of ocular movements varies greatly in different animals; vestibular stimulation which causes rotation of the human eyes in the frontal plane, produces in fishes merely an upward deviation of one eye and a downward deviation of the other. Caloric stimulation of cats fails to produce some of the responses with which we are all familiar in the human being.

One of the most reliable methods of tracing the course of nerve fibers is by means of the degeneration which follows their injury. Such degeneration can be traced only in the distribution of the particular fibers injured. Although, physiologically, nervous impulses pass from the terminals of one nerve fiber to another neuron, and so on to their ultimate destination, pathologically considered, each synaps limits further degeneration. To repeat, degenerations can only be traced within the limitations of a single neuron—always being limited by the synaps. Many different pathways, such as that of common sensation from the skin, are known to include three or more successive neurons, including as many synapses through which the impulse must pass. To follow this pathway by the process of tracing degenerations, it is necessary to produce a lesion in each successive neuron. For example, the sensory pathway for pain extends from the periphery to the posterior root ganglion; other fibers pass from there and enter the spinal cord; a short distance above the point of entry, connection is made with another neuron, the axis cylinder of which proceeds up the cord and through the brain-stem to the optic thalamus. Here in turn, new connections are made; the ultimate anatomic connections are as yet undetermined. The different forms of sensation, such as heat, cold, tactile, pressure and muscle-sense, are each represented by a special neural pathway which can be traced over successive neurons along its course. Some proceed up the spinal cord on the same side that they enter, some

on the opposite side; some are interrupted within the cord shortly after their entry, whereas others proceed without interruption to nuclei in the medulla oblongata.

Analogous arrangements may be noted in the nervous mechanism of the special senses. The retina, for example, contains within its layers not only the end-organs, represented by the rods and cones, but neurons interposed between these end-organs and the neurons which proceed in the optic nerve. Furthermore, the primary centers of vision form another interruption in the course of the visual pathways before they reach the cerebral cortex.

There are only a few demonstrated synapses along the vestibular pathways, such as Scarpa's ganglion and Deiters' Nucleus; however, it is fair to assume from the analogies just given, that there are numerous other breaks in the continuity of the neural vestibular pathways.

From the well established principles of secondary degenerations, it is readily seen that any part of any pathway can be traced only within the segment which has been injured, and that to trace any particular pathway throughout its various segments, it would be necessary to produce lesions such as to involve each segment in turn.

In applying this method to the present problem, there arises the practical difficulty of making discrete lesions. To be of value, experimental lesions must be limited so as to destroy a small amount of tissue, and must be so located as to produce the desired destruction without disturbing adjacent tissues. In vestibular studies, it would be highly desirable to produce such minute lesions within Deiter's, von Bechterew's and the Triangular Nuclei; the cerebellar nuclei, globosus, emboliformis, and festigii; the corpora quadrigemina and many other structures. The difficulties attending such attempts are obvious.

To give concrete applications of the above general principles, we might consider the possible results to be obtained by destruction of successive segments in the vestibular pathways—

(1) End-organ, (2) VIII Nerve and (3) Central Nuclei.

1. End-organ. The exact histologic course of fibers from each semicircular canal could be demonstrated if one canal could

be destroyed and its pathways traced. (The same could be applied to the utricle and saccule.)

It is possible to destroy one horizontal canal and its ampulla, in an animal previously proven to be normal by caloric stimulation. To prove that only the horizontal canal has been destroyed, it would be necessary subsequently to demonstrate normal function in the vertical canals. This proof is probably impossible because of the serious disturbance of the entire mechanism of the end-organ incident to the destruction of one canal. This would seem to be corroborated by the experiments of Sachs and Alvis, in the lack of uniformity of the spontaneous nystagmus which they observed, in the different animals in which only one canal had been destroyed.

Even if one canal could be destroyed and the fact of this isolated destruction proved by the caloric test, the secondary nerve degeneration does not extend throughout the physiologic distribution but stops by Scarpa's ganglion. Sachs and Alvis have demonstrated that destruction of all the canals is not followed by degeneration in the VIII Nerve.

2. The VIII Nerve can be destroyed and secondary degeneration traced to different portions of the brain-stem and cerebellum. However, it has not yet been possible to determine any relationship between any portion of these fibers and the individual semicircular canals, or the utricle or saccule.

3. Central Nuclei. Isolated destruction of the central nuclei is much more difficult; and yet, to correlate the different nuclei with the different portions of the end-organ, such a method seems to offer the only possibility for future study. In the work already done, in making discrete lesions in the nuclei of the brain-stem, so far as we know only the spontaneous phenomena have been observed. Additional information might be gained not merely by observing the spontaneous phenomena but by studying the responses to the caloric test.

If, for example, Deiter's Nucleus were destroyed and subsequently the horizontal canal failed to give normal responses whereas the vertical canals did produce normal responses, we could reasonably conclude that the fibers from the horizontal

canal go to Deiter's Nucleus and that the vertical canals fibres do not go to Deiter's Nucleus. So with each of the other vestibular nuclei—the von Bechterew's and Triangularis—if it ever becomes possible to make discrete lesions of these structures.

Summary: To find out the exact course of the fibers from the different semicircular canals appears to be impossible from experimental work on the end-organ or VIII Nerve. If ever this is to be attained, it seems that the only possibility consists in making discrete lesions in the brain-stem and then testing the different semicircular canals by the caloric test.

CLINICO-PATHOLOGIC RESEARCH.

Clinico-pathologic study has its own peculiar advantages, among them:

1. The human being is the object of study. This eliminates certain fallacies always present in animal study. Studies of the human being are furthermore more complete, in that it is possible to secure the co-operation of the patient in making more elaborate tests than are possible in animals. There is another important advantage in that the human being can supply intelligent evidence of his subjective sensations, both spontaneous and after experimental tests.

2. Certain pathologic processes result in small discrete lesions, in locations within the central nervous system, which can not be duplicated by operative work on animals. This includes vascular lesions, such as thrombosis and small foci of encephalitis and multiple sclerosis. Incidentally, lesions of this type give more exact information than tumors or abscesses, in which pressure phenomena may obscure the picture.

3. Clinico-pathologic study has the advantage of having its workers constantly pressed by the need of accuracy in diagnosis. Every clinician feels the necessity of accurate interpretation of phenomena which he observed in every day practice. There may be, in the world, a dozen or a score of experimental investigators interested in neuro-otology. In contrast, every otologist and every neurologist is a potential investigator in clinico-

pathologic research. Furthermore, problems have been raised by clinical studies which have stimulated experimental anatomic studies. The ideal, of course, is to obtain exact histologic knowledge of each minute detail. In the meantime, the clinician can serve two purposes: first, to learn what he can from clinical cases, or operation or necropsy; and second, by his daily clinical observations to raise questions and present problems for the anatomist to try to solve.

In discussion experimental work on animals, it has already been mentioned that normal responses to ear-stimulation should be made a basis of observations. It may be said that clinical experience with human subjects has already fairly established the norm. We know, with hardly any reservation, that when any normal individual is turned or his ears douched, there is produced a definite eye movement of certain duration, in a definite plane; a definite vertigo of a certain duration, and a falling in a definite direction. If he be turned or douched to an extreme degree, he shows pallor, sweat and nausea.

With the above phenomena we become familiar, and all who undertake such tests become impressed with the uniformity of responses.

Each variation from the norm must receive due consideration. To be sure, a certain latitude must be given to individual variations within the norm; when, however, gross deviations from the norm present themselves, it may be concluded that some portion of the complicated mechanism has been disturbed. It might be well to cite briefly certain examples of clinico-pathologic experience in patients that proved to have intracranial lesions.

1. In one patient, ear stimulation produces normal responses from the horizontal canals and no responses from the vertical canals. Another patient shows the reverse—normal responses from the vertical canals and absent responses from the horizontal. Other cases, such as angle tumors, have shown no response from any canals of either ear, except one horizontal canal, and this showed entirely normal function. Two cases recently seen (Exhibit A and Exhibit B), not as yet pathologically verified, showed normal responses from all semicir-

cular canals, except one horizontal—the exact opposite of the phenomenon-complex of the angle tumor. We conclude that there is necessarily a neuraxial differentiation—that the fibers from the horizontal canal are sufficiently separated from those from the vertical canals to permit either to be involved without any involvement of the other. This will be taken up in detail under the separate “Problems”.

2. Comes a patient who shows normal nystagmus, but impaired vertigo on stimulation of one horizontal canal. Another shows normal nystagmus and impaired vertigo from both horizontal canals. Another shows normal vertigo but impaired nystagmus. We are led to consider that from the horizontal canal, there is a further division of the pathways, on the one hand producing the nystagmus and on the other hand producing the vertigo.

Clinico-pathologic observations of lesions in different intracranial locations have shown such different phenomena that to explain them merely on the basis of pressure from internal hydrocephalus is untenable.

In spite of the limitations in experimental and in clinico-pathologic research, each method has its own peculiar advantages and in many ways supplements the other. Further progress will be determined largely by the measure of co-operation of the investigators using the different methods of approach.

IV. PROBLEMS.

To the clinician the problem is to determine the valuation to be placed upon each phenomenon observed in the study of patients. We will take up separately a number of debated subjects and will present, in skeleton form, certain evidence against and for previous conceptions of their significance. It will thus be possible for the discussion to round out our knowledge concerning these separate problems. Realizing that there are many doing neuro-otologic work that would not be present at this meeting, we also sent 80 questionnaires.

It will be noted that the subject of forced movements of the skeletal musculature is not included in these problems. Magnus

and de Kleijn and others have demonstrated physiologically that there is a lower circuit from the internal ear to the skeletal muscles—presumably via the vestibulo-spinal tracts. It would seem that these vestibular impulses are eventually distributed by spinal co-ordinating mechanisms in control of the musculature affected. In this sense, the vestibulo-spinal pathway is analogous to the vestibulo-ocular pathway. As to past-pointing, as tested by the Barany method, although it is essentially an adjustment to the conscious sense of movement, yet it is probable that tonetic influences through the lower pathways reinforce this adjustment. The falling response to ear stimulation appears to be essentially a forced movement, although there is a definite cerebral element. There is, however, so much uncertainty as to the clinical interpretation of forced movements that we have not attempted to present it as a problem.

The problems will be presented in the following order :

1. Auditory.
2. Endolymph movement.
3. Mechanism of ocular movement.
4. Concept of vestibulo-ocular pathways.
5. The quick component of nystagmus.
6. Spontaneous vertical nystagmus.
7. Perverved nystagmus.
8. Vestibulo-cerebello-cerebral tract.
9. Cerebello-pontine angle phenomenon-complex.
10. Neuraxial differentiation of pathways from the different semicircular canals.

AUDITORY.

This subject offers nothing in the form of an argument against or for any existing conceptions. The anatomy, physiology, pathology and symptomatology of the cochlea and the

auditory portion of the VIII Nerve are well established. The cortical cerebral areas for the reception of auditory impulses have been demonstrated to be in the first temporal convolutions; this may be considered as having been rather conclusively determined, both by experimental work on monkeys and by clinico-pathological cases.

However, as to the course of the pathways between the cortical centres and the entrance of the VIII Nerve into the brain-stem, clinico-pathologic evidence is singularly meagre. A priori, it might be expected that various brain-stem lesions would exhibit an impairment of hearing. Observations of lesions within the brain-stem without hearing impairment are common. In fact, hearing impairment from brain-stem lesions is so uncommon as to be almost unknown in the experience of otologists. The generally accepted pathway of the auditory impulses is as follows: From the auditory nuclei by way of the lateral fillet to the corpora quadrigemina and internal geniculate in both direct and crossed relations; from this location the pathway continues to the first temporal convolution of each cerebral cortex. For many years no new evidence on this subject has been presented and we rely on the uncontradicted work of the older anatomists and pathologists. The explanation advanced for unimpaired hearing in cases of brain-stem lesions, is the neuraxial differentiation of the auditory fibres, by means of which each VIII Nerve is in relation with both sides of the brainstem. The auditory pathways on each side are made up of fibres in relation to each cochlea. In other words, with this arrangement of fibres, deafness in either ear cannot be produced by a lesion on either side of the brain-stem or cerebral pathways, but would have to involve these pathways on both sides. The absence of impairment of hearing from unilateral lesions of the supranuclear pathways is however noteworthy. Some evidence of impaired auditory function might well be expected in brain-stem lesions. It may be that our methods of testing auditory function might be improved in order that we might detect such a type of impairment. In the present status of our knowledge, however, auditory tests are of practically no value in giving evidence of lesions within the brain-stem.

Attention might be called to a separate neuro-mechanism concerned with the peripheral organ of hearing; this concerns the mechanism controlling the tensor tympani and stapedius muscles.

These two muscles are so arranged that they appear to be concerned with the adjustment of various degrees of tension of the drum membrane and the ossicles. These adjustments are made to accomodate the middle ear mechanism to various degrees of sound waves. The tensor tympani muscle is supplied by a branch of the motor division of the V Nerve; the stapedius muscles by a branch of the VII Nerve. In many respects the neuro-motor apparatus controlling the adjustment of these muscles is analagous to the accomodation mechanism of the eyes, in which the ciliary muscle innervated by the III Nerve, automatically controls the focus of the crystalline lens, to maintain clear vision.

So far as we know the action of these muscles is automatic. That there is a lower mechanism, presumably within the brain stem, and that a cerebral control can be exerted on this lower automatic mechanism is suggested by the following. One of the writers is able at will to produce a roaring in both ears. Try as he will, he cannot produce this roaring in only one ear; in fact he cannot make one ear roar louder than the other. This sound, produced by voluntary effort, consists of a rapid sequence of beats. There is a definite intermission between those beats. The beats and the intermissions are synchronous in both ears. There appear to be perhaps fifteen to the second; this is of course merely approximate, but there is a definite rythm. The slightest effort produces this roaring, at any time; it is however a definite effort to maintain the roaring. By concentrated effort the ears can be made to continue to roar for 18 seconds; the roaring then ceases, but can be made to recur at once after cessation. It would be difficult to explain this matter on any other basis than that of a voluntary control over the action of either the tensor tympani or the stapedius muscle, or both.

ENDOLYMPH MOVEMENT *

The first problem of the vestibular apparatus is that of the

physics of the end-organ. Upon theoretical considerations it has been generally assumed and regarded as more or less determined, that the endolymph moves freely throughout all portions of the membranous labyrinth; that the endolymph movement in the semicircular canals acts upon the nerve receptors in the crista ampullaris of each canal; and that the direction of the endolymph movement determines the nature of the afferent impulse. The peculiar physical structure, and the configuration of the semicircular canals and utricle and saccule lends credence to this view. Given an organ made up of open tubes, placed in definite planes, and containing endolymph throughout their full extent, it has been thought obvious that this sense-organ registered changes of position of the head by means of mass movement of the contained fluid. Doubt has been expressed as to the accuracy of this theory, especially concerning the possibility of endolymph movement within the membranous semicircular canals.

Question:

Does the endolymph move freely within the membranous labyrinth?

Against:

Physical considerations.

1. It would seem that the small calibre of the membranous semicircular canals must interfere with mass movement of the lymph within them, on account of capillarity.

2. It would seem possible that pressure variations, without mass movement of endolymph, can explain the origin of impulses from the end-organs.

Physiologic considerations.

1. In fishes in which the semicircular canals and their ampullae had been destroyed, S. S. Maxwell has produced all normal eye responses, except the horizontal, by mechanical stimulation of the otoliths of the utricle. This is an important contribution to the physiology of the internal ear. He further showed that the horizontal canal can be ligated or elevated into the vertical position, without interfering with the responses

from turning in the horizontal plane. He points out that all the essential responses can be produced under conditions in which endolymph movement is impossible.

For:

Physical considerations.

1. The physical arrangement of the semicircular canals is theoretically ideal for recording inertia of the contained fluid, from every possible movement of the head.

2. Maier and Lion (*Arch. f. Ohren, Nasen u. Kehlkopfheilk.*, 107:149, Leipzig, May 20, 1921) have observed movement of the endolymph within the semicircular canals of living animals, after caloric stimulation.

3. One of us has demonstrated mass movement of fluid within a capillary glass tube under conditions approximating those of the semicircular canals. The lumen of the tube is between .3 and .4 mm.; the tube is semicircular and both ends terminate in a small reservoir or artificial utricle. The entire apparatus is filled with vinegar, which contains particles in suspension, easily observed under the microscope. With this tube in a verticle position, and the reservoir uppermost, raising or reducing the temperature of one side is followed by circulation of the liquid which can be easily observed under the microscope. The movement of the liquid begins slowly and gradually increases to a maximum velocity which is maintained as long as the conditions of temperature in the tube are maintained. The movements of the liquid are always in the direction corresponding to the principles of the thermo-syphon. During one observation of these caloric tests, one end of the tube accidentally became blocked; when heat was applied near the opposite end, two currents in opposite directions were observed at the same point within the lumen of the tube. In other words, free fluid movement was demonstrated in two different directions within a tube of the calibre of .4 mm. or less.

The above physical experiment would seem to show that capillary resistance does not prevent mass movement of fluid within a capillary tube, when the fluid is in free communication with a common reservoir through both ends of the tube.

Physiologic considerations.

1. It is perhaps possible to explain Maxwell's experiments without sacrificing our faith in the old tenets of endolymph movement. He has emphasized the importance of the otoliths, about which so little is known; and he has shown that certain eye movements can be produced in fishes, irrespective of endolymph movement within the semicircular canals. This, however, is not positive evidence that, under normal conditions, the endolymph does not move.

The eye movements in fishes, upon which these observations are based, do not compare in variety or delicacy with the responses elicited in the human being. When the horizontal canal of the fish was ligated, it is possible that the impact of the fluid acted on the sensory nerve-endings in the ampulla, even though this movement could not continue throughout the entire course of the canal. A gross horizontal movement of the eyes could be obtained under these conditions; this, however is qualitative, and not quantitative. If after ligating the canal, there could be produced an after-turning nystagmus of normal duration and amplitude, and after the caloric test a full response of normal intensity and duration, we would then have more positive evidence against endolymph movement.

Again, when the horizontal canal was elevated to the vertical position, it is not probable that the ampulla was drawn out of place because it is directly adjacent to the utricle.

2. The argument that tension, and not endolymph movement, determines the response is not tenable; a change of tension without movement is impossible. If after turning, there is a tension at one end of the utricle and a relaxation at the other end of the utricle there must have been a displacement of the fluid content—in other words, mass movement.

3. In cases of fistula into a semicircular canal, pressure applied produces *immediate* responses. These responses *cease promptly on the cessation of the pressure*. There is evidently no prolongation of the response after the stimulation has ceased. After turning, the nystagmus and vertigo normally continue from 20 to 30 seconds. From this it may be concluded that the stimulus has persisted from 20 to 30 seconds after the turning

has ceased. Immediately after turning, the nystagmus and vertigo are at their maximum. They gradually subside; the amplitude of the nystagmus becomes smaller and smaller and the individual feels that he is rotating slower and slower, until he comes to a full stop. This is a common observation and can be demonstrated on any normal individual; it would seem that it can only be interpreted on the basis of a movement of the endolymph, which is at its maximum immediately after the turning and which gradually diminishes until it comes to a full stop.

4. A recent pathologic case bears directly on this subject. (Exhibit C., Georgie Johnson).

In the examination of this patient, pneumatic pressure in the left ear produced:

1. Nystagmus, large amplitude, horizontal; slow component to the right; quick component to the left.

2. Past-pointing of all extremities to the right.

3. No falling—all responses were in the horizontal plane.

The nystagmus and past pointing appeared promptly when pressure was applied; continued while pressure was maintained; and ceased at once when pressure was discontinued.

These responses fulfill the conditions of the fistula test and indicate a fistulous opening into the left horizontal canal.

In contrast to the active responses from the fistula test, the left horizontal canal gave no response to turning or caloric stimulation.

Obviously the crista ampullaris of the left semicircular canal, with its sensory receptors, was functioning. The responses from its stimulation were prompt and qualitatively correct. They were elicited not only by air pressure, but also, as shown in the motion pictures, merely by touching the fistula region lightly with a cotton-tipped probe.

It is of particular interest that, although the crista ampullaris was actively functioning, yet it was entirely unaffected by turning and douching. Obviously, something interfered with the function of the horizontal canal itself. The most reason-

able explanation of these phenomena appears to be that there was an obstruction in the horizontal canal, posterior to the fistulous opening, which interfered with the endolymph movement within the canal, and which did not permit endolymph movement or impulse to affect the ampulla.

In Ewald's original experiment on pigeons, he plugged the horizontal canal just behind an opening in the canal. Pressure caused the eyes to rotate toward the opposite side. This case of Georgie Johnson closely resembles this experiment of Ewald. A similar case was shown by one of us before this Society in 1917—case of John Brooks. It happened that the fistula in this case was in the *right* ear and showed:

1. Nystagmus large amplitude, horizontal; slow component to the left; quick component to the right.

2. Past-pointing of all extremities to the left.

3. No falling—all responses were in the horizontal plane. These two cases, Georgie Johnson and John Brooks, show all the phenomena of horizontal canal stimulation, by means of the fistula test. Johnson showed the normal responses to be expected on stimulation from behind the left ampulla, whereas Brooks showed the diametrically opposite responses to be expected on stimulation from behind the right ampulla. No positive deductions should be made from either of these cases; the only positive proof would be complete histologic examination, demonstrating an obstruction in the horizontal canal, posterior to the fistulous opening. Short of histologic proof, however, these cases seem to be exactly analagous to the original Ewald experiment.

Resumé:

Considering the available evidence, the following conclusions are suggested:

1. Capillarity does not prevent endolymph movement within the semicircular canals.

2. Endolymph movement within the semicircular canals is an important factor in determining the character of the impulses from the end-organ.

3. The otoliths play an important part in the detection of movement; apparently the otoliths and cristae of the semi-circular canals cooperate in function.

MECHANISM OF EXTRAOCULAR MOVEMENT

The preceding problem concerned the physics of the end-organ and the mechanism of the production of stimuli; the next problem is that of eye movement resulting from ear stimuli. Before attempting, however, to consider the vestibulo-ocular mechanism, it seems wise to discuss the ocular mechanism with which the ear is connected. We will therefore attempt to analyze the essential mechanism of ocular movement itself; then to follow the functional relationship that exists between the internal ear mechanism and this ocular mechanism. Perhaps such a study of this functional relationship, correlated with historical experimental studies, may lead to an advanced conception of the anatomic relationship.

In the more primitive stages of the evolution of the ocular mechanism, one eye is placed on each side of the head and covers its own individual field of vision. In this stage each eye may be moved independently. In higher forms, the two eyes are placed anteriorly, and to a large extent cover the same visual field. The fields are thus necessarily superimposed, and fusion of images is attained. As binocular single vision, normal to higher animal types, necessitates maintaining fusion in all possible directions of gaze, an accurate adjustment of the visual axes must be maintained. Thus is evolved the conjugation of ocular movements—an automatic process. The term, "associated ocular movements," signifies the unity of purpose of the two eyes which operates in such a manner as constantly to direct both visual axes toward the fixation point in all possible directions of gaze. This association of movements is not only automatic and involuntary, but is so firmly established as to defy voluntary effort to avoid it. No normal individual can voluntarily rotate one eye in any direction in the slightest degree without also rotating the other.

By the very nature of the anatomic relations of the eyeballs and the extraocular muscles, it may readily be seen that *each of*

the twelve extraocular muscles must play a definite part in every normal ocular movement. In each movement, each muscle acts either as prime mover or synergist or control. These twelve muscles can instantly and with micrometric accuracy direct the two visual axes toward any point within the range of the possible eye movements. This is true not only as regards direction of gaze but also as regards distance. The accurate degree of convergence of the axes to correspond to the distance of the fixation point is a part of the adjustment.

What is the nervous mechanism involved in extraocular movements? Manifestly this involves at least three segments, viz: (1) The peripheral or neuro-muscular mechanism; (2) The cerebral projection tracts, and (3) An intermediate or co-ordinating mechanism.

1. The peripheral or neuro-muscular mechanism comprise the III, IV and VI Nerves and their nuclei, and the extraocular muscles which they supply. The nerves of this mechanism constitute the final common pathway for all impulses causing ocular movement. As previously indicated, this entire mechanism is called into action for each eye movement, whether voluntary or reflex.

Disturbance of any portion of this mechanism results in a porportionate loss of movement in terms of individual muscles or nerves, e. g. paralysis of the abducens, or paralysis of the III Nerve.

2. The cerebral projection pathway, by which voluntary ocular movement is accomplished, is commonly belived to be within the pyramidal tract.

It should be mentioned that fibers of the pyramidal tract have not been histologically traced directly to the cells of the eye-muscle nuclei. It should also be noted in this connection that pyramidal fibers have not been traced to the motor cells of the spinal cord, or indeed to any ultimate destination. From this we may gain some idea of the difficulties which beset histologic demonstration of a nerve pathway even though its existence be proven by physiologic observation.

From the viewpoint of the cerebral motor mechanism, the eye movements conform to the general rule applying to all bilaterally grouped voluntary movements. They are not paralyzed, and are but slightly affected by cerebral lesions which cause hemiplegia; either cerebral hemisphere is apparently capable alone of calling into action the complete range of normal extraocular movements. The usual interpretation assumes that there is a bilateral cortical representation for all muscles comprising such muscle-groups. Other examples of similarity grouped movements are: wrinkling of the forehead, phonation, respiration and trunk movements. All of these show but little involvement in the hemiplegic state.

Conjugate deviation of the eyes may result from acute unilateral cerebral lesions or irritation, usually in association with convulsions or disturbance of consciousness, but this conjugate deviation never persists as a permanent symptom. Cerebral lesions do not interrupt the conjugation of eye movements. This is true even following decerebration, if the mid-brain is not injured.

3. The coordinating mechanism of ocular movement. The peripheral neuro-muscular mechanism is well determined. The cerebral projection pathway is commonly accepted. However, to explain the phenomena of extraocular movement, it is necessary to postulate a coordinating neural mechanism, interposed between the cerebral and the peripheral segments. It is this coordinating mechanism that commands our particular attention. The intricacies of this structure have so far precluded exact knowledge; but its existence is proven by the following evidence.

In describing the phenomena observed in their experiments on dogs and cats, Wilson and Pike, (*Phil. Tr. Roy. Soc. London* 803:127-160, 1912) noted conjugate ocular movements produced by ear stimulation, (electric and caloric), in animals after decerebration. These eye movements were in lateral and oblique directions. Similar reflex conjugate movements were recorded after removal of the cerebellum,

In one of our experiments on cats, conjugate eye movements were observed after decerebration. A transverse incision was made anterior to the tentorium; necropsy showed that this incision had been carried to the base of the brain and that the cerebral hemispheres had been separated from the brain-stem, with exception of a part of each occipital lobe. Caloric stimulation of the right ear produced a few horizontal nystagmic eye movements; the amplitude was small and only a few movements were elicited, but the eye movements were definitely conjugate. After the head was removed from the body of the animal, electric stimulation, applied to the region of the internal ear of each side, caused conjugate vertical ocular movements.

The significance of these observations is in the persistence of the conjugation of eye movements after interruption of the cerebral or cerebellar connections. The type of movements observed requires the concerted, coordinated action of all the extraocular muscles. Since the cerebrum and cerebellum can be excluded, there remains only the brain-stem in which to look for this coordinating mechanism. It would seem that this mechanism must include a system of neurons with fibers distributed to all the cells of the peripheral motor nerves; also that this mechanism is capable of sending to all these cells, the correct impulses necessary to produce any accurately coordinated conjugate eye movement. This is also indicated by the variety of reflex eye movements observed after vestibular stimulation. It would seem probable that this coordinating mechanism not only receives afferent stimuli from various peripheral sources and directs the appropriate reflex responses, but that it is only by way of this mechanism that impulses reach the eye muscles, from the cerebrum or any other source.

Clinico-pathologic observations furnish important evidence concerning this mechanism. Many cases on record have shown inability to rotate the eyes in certain directions of gaze, although there was no paralysis of any of the extraocular muscles. These cases are designated as paraly-

sis of associated ocular movement to the right, or to the left, or upward, or downward, or in convergence. Such cases show lesions involving the posterior longitudinal bundle or the brain-stem near the aqueduct of sylvius. Cases of this type, showing a disturbance of associated eye movements without paralysis of any of the eye muscles, point definitely to lesions of the mechanism under discussion, and indicate that the posterior longitudinal bundles and other association and commisural tracts within the brain-stem are utilized by this mechanism.

According to the above conception, all eye movements, whether voluntary or reflex, are under the control of this coordinating mechanism. It would seem that this mechanism not only acts as a switchboard of distribution but also manifests a certain energy derived from its own neurons—similar to that of the electric amplifier. This energy is distributed in a selective manner and results in a conjugate movement. To cite an analogy in military manoeuvres: the two eyes, being analagous to a squad, execute "squads right" or "squads left" at the order of its leader; the leader moves his squad as a unit at the command of the company commander. If the commander is killed the leader still controls his squad as a unit. If no order be received by the leader, the squad may wander about aimlessly—still as a unit—and under the leader's direction. The squad leader in this analogy is the coordinating mechanism in the brain-stem.

The classic extraocular symptom of lesion of the peripheral neuro-muscular mechanism, is paralysis of individual ocular muscles; of the cerebrum, is conjugate deviation; of the coordinating mechanism of the brain-stem, is disturbance of associated eye movements.

Many other coordinated neuro-muscular mechanisms might be cited as analogies for this conception of a coordinating eye mechanism. For example, the respiratory movements are controlled by the respiratory center. All the movements of respiration are conducted by voluntary muscles, each supplied by peripheral motor nerves; the

whole series of these nerves arises from different levels, from the pons down to the lower dorsal segments of the cord. The respiratory center functions automatically through the influence of CO_2 in the blood; the automatic action of this center keeps up continuously throughout the life of the individual, whether asleep or awake. However, voluntary control through cerebral impulse, may step in and modify respiratory movement, by accentuation, change of rhythm or temporary arrest. Reflexly, various afferent impulses may also step in, such as application of sudden cold to the skin and emotional reactions. It must be noted, however, that whatever impulse is transmitted to the respiratory center, the response is in terms of synchronous action of many muscles controlling the respiratory movements. Decerebration or removal of cerebellum does not interfere with the automaticity of the respiratory mechanism, or with the harmonious action of the muscles it controls.

Coughing, sneezing and vomiting are controlled by similar coordinating mechanism or nervous centers each one of these centers serves its own definite purpose, and each center controls its own definite group of muscles to meet a specific physiologic need.

In this connection it is instructive to consider how few are the fibers of the cortico-spinal or pyramidal tract compared with the much greater number of peripheral motor neurons which they control. This signifies that each fiber of the cortico-spinal motor pathway controls a number of peripheral motor neurons through the intermediate neurons of a coordinating mechanism.

It must not be thought that the actual structure or content of these centers has been fully determined. The respiratory center is regarded as being situated in the medulla oblongata at the apex of the calamus scriptorius and is supposed to be comprised of cells located in the reticular formation on both sides of the midline. There is no unanimity of opinion, however, as to just what neurons are actually included in the formation of this center. The same

may be said for all the other coordinating centers. Their existence is unquestioned; their action, in a large measure an independent action, has been repeatedly demonstrated by experimental work as well as clinical observation; and yet we must not lose sight of the fact that their exact structure is unknown.

Summary:

Evidence indicating the existence of a coordinating mechanism, consisting of intermediate neurons situated between the cortico-medullary and peripheral motor pathway:

1. The complete conjugation of the eyes in all normal voluntary and reflex movements.
2. Conjugate ocular movements can be elicited by either cerebral hemisphere.
3. This conception of the coordinating ocular mechanism is in accord with other well-known coordinating mechanisms, such as the respiratory center.

Evidence indicating that this coordinating mechanism is located in the brain-stem:

1. Conjugate ocular movements can be elicited through vestibular stimulation, in animals, after removal of cerebrum or cerebellum.
2. Cases of paralysis of associated ocular movement have shown lesions in the brain-stem.

From the above consideration of the coordinating mechanism itself we now turn to a discussion of its functions; how it is brought into action by stimuli from the cerebrum and from various sense-organs.

Voluntary movements of the eyes, under normal conditions, consist of associated movements in all conceivable directions, except rotation in the frontal plane, and divergence. In close relation to the voluntary movements are certain instinctive automatic movements, in the production of which, a cerebral element is evident. Example: turning the eyes in the direction of objects suddenly coming into

the peripheral field of vision; maintaining fixation of vision upon moving objects; rotation of the eyes in the frontal plane, which normally occurs when the head is tilted to one side; the necessary degree of convergence to attain fusion for objects at different distances from the eyes; rotation of the eyes in the direction from which sounds are heard; or from which a touch is felt. This type of movement occurs instinctively, swiftly and accurately; without conscious effort, and occurs in the interest of securing visual efficiency. Conjugation of ocular movement is always maintained.

These instinctive automatic movements tend to approach the purely reflex. In fact it has been suggested by some that visual, auditory and tactile impressions may initiate ocular movements through the lower centers directly. There is some evidence that a direct reflex action can occur, but it is difficult to eliminate a cerebral element as a probable factor in most reflex ocular movements. There is, however, at our command one means of demonstrating a direct reflex movement of the eyes through the lower pathways; by vestibular stimulation in decerebrate animals.

CONCEPT OF THE VESTIBULO-OCULAR MECHANISM.

Up to this point we have discussed the essential mechanism of extraocular movement. We are now in position to discuss the functions of the mechanism in terms of its reactions to stimuli from the internal ear.

Regardless of the exact physics of endolymph movement, the eye movements resulting from the turning and caloric tests are always in the exact plane of the endolymph impulse; further, the direction of the slow component of the nystagmus is always in the direction of the endolymph impulse. The almost mathematical precision of these reactions is familiar to all observers, irrespective of what theory is advanced to explain them.

Electric stimulation of the end-organ, although a delicate test of function, is general and not selective. The caloric test is selective, but to a limited degree. The caloric effect

can be applied only from the lateral aspect; therefore the internal ear can not be influenced in sagittal or oblique planes. It is the turning test that not only involves both ears but stimulates them in a manner identical to their daily physiologic activity.

The turning tests can be made to influence the internal ears in any plane desired. In this way, an infinite gradation in the nature of the impulses can be sent inward from the end-organ, depending only on the position of the head during the turning. Each gradation calls forth its own specific movement of the eyes.

That a reflex associated movement of the eyes in any pre-determined direction or plane can be elicited, at will, by appropriate ear stimulation, implies an accuracy of adjustment and intimacy of neural connections of the utmost variety and nicety. We must postulate not only an accurate gradation of end-organ impulses, but a like series of adjustments of the ocular coordinating mechanism. In each adjustment, all twelve ocular muscles are concerned in an alternating series of actions and reactions.

Such phenomena can be explained only by the concept, that impulses from the internal ear go to the ocular coordinating mechanism, which determines the appropriate ocular movements. By this concept, fibers from the vestibular nerve and nuclei are not distributed to the peripheral motor neurons of the III, IV and VI cranial nuclei, but to the ocular coordinating mechanism. This would explain the failure of attempts to trace the vestibulo-ocular tracts to the eye-muscle nuclei, by means of secondary nerve-degenerations.

THE QUICK COMPONENT OF NYSTAGMUS.

Question:

Is it a cerebral function?

The usual concept of vestibular nystagmus has been that the quick component is of cerebral origin. There seems to be no question as to the origin of the slow component; but

there has been nothing conclusive concerning the origin of the quick component.

For:

The concept of the cerebral origin of the quick component is suggested by evidence such as

1. Voluntary fixation is a cerebral act. Deviation from the point of voluntary fixation occurs when the ears are stimulated. The explanation has been suggested that the quick component is an expression of the cerebral influence in momentarily overcoming the reflex pull from the ear.

2. During a vestibular nystagmus, gaze in direction of the quick component amplifies the nystagmus, whereas gaze in the direction of the slow component reduces it, even to the point of complete disappearance. It seems that it is optional with the cerebrum to determine a maximum or minimum nystagmus, by the simple expedient of voluntarily reinforcing either the quick or the slow component.

3. Loss of the quick component under general anesthesia; also under other unconscious states, such as coma.

4. Case of Miss C. (Equilibrium and Vertigo, P. 382). On caloric stimulation of right vertical canals, there occurred a rotary conjugate roll of both eyes toward the right, no quick component to the left; stimulation of right horizontal canal produced conjugate deviation to the right, which persisted for the duration of the stimulus without any quick component to the left. Stimulation of the left vertical canals produced a normal rotary nystagmus to the right. Stimulation of the left horizontal canal produced normal horizontal nystagmus to the right. The contrast was definite; the quick recoil to right was present and the quick recoil to the left was absent. Necropsy a few days later showed a large subcortical abscess in the right parietal region.

Against:

In contrast to the above, the following arguments make it strongly suggestive that the quick component can be

initiated by a lower mechanism, independent of the cerebrum.

1. The first argument given above, (that the quick component is an attempt at cerebral fixation), is minimized when we note that the quick component is no less active when visual fixation is eliminated—by closing the eyes. Further the intimate reciprocal relationship between the slow and quick components would seem to indicate the operation of a single mechanism, rather than the cooperation of widely separated structures. On the basis of a cerebral origin of the quick component, we would have to consider the following sequence; ear stimulation would act for the fraction of a second, whereupon a cerebral impulse would inhibit that from the ear and then substitute one of its own, effecting a reverse movement. This cerebral impulse, quickly subsiding, is then replaced by that from the ear and this alternating series of reverse impulses continues for the duration of the stimulus from the ear. This appears less reasonable than a concept of the operation of a lower coordinating mechanism.

2. Absence of the quick component in general anesthesia or other unconscious states by no means proves its cerebral origin. The different degrees of disturbed consciousness are accompanied by the loss not only of cerebral activities, but also by the loss of the various lower reflexes at different stages of the anesthesia.

3. Numerous cases of lesions in the cerebrum, including hemiplegias, temporal lobe lesions and occipital lobe lesions have failed to show loss of quick component.

4. Many cases of proven posterior fossa lesion, including cerebellar tumors and lesions of the brain-stem have shown absence of quick component. (Cite case of cerebellar abscess—Castillo boy, Exhibit D.)

5. Certain cases of pathology of the brain-stem in which ear stimulation produced dissociated nystagmus—nystag-

mus of one type in one eye and of a different type in the other eye. It would be difficult to explain these two different types of quick components on a cerebral basis. When one sees, after ear stimulation, a horizontal nystagmus in one eye and a vertical nystagmus in the other eye, it seems more reasonable to conclude that the quick component originates in a lower mechanism which automatically reacts to correct the particular deviation of each eye.

6. This conception of a lower automatic center which can produce the quick component, independent of the cerebrum, seems to be supported by recognized analogous coordinated reflexes. If acid is applied to the side of a decerebrate frog, the hind leg of the same side is moved in a manner to wipe off the irritant. This movement is not simply a flexion or an extension of the leg, but an orderly sequence of movements including flexion and extension of several joints in a manner to accomplish a definite purpose. If the back of a decerebrate dog is rubbed, the so-called "scratch reflex" may be elicited, consisting of a series of quick to and fro scratching movements of one hind leg. If a stream of cold water is directed against the inner surface of the pinna of a decerebrate cat, one may observe a quick shaking to and fro movement of the head which is characteristic of normal cats in attempting to dislodge irritants from the ear.

In an analysis of the movements concerned in such coordinating reflexes in decerebrate animals, it is particularly to be noted that there is in each reflex a series of reciprocating to and fro movements, performed by groups of muscles. Many muscles are concerned in each reflex, and different groups are active in different phases of the movements, alternating with remarkable accuracy and rhythm. These reflexes are of the nature of purposeful movements, and each of them can be initiated by volition. They have probably evolved from purely voluntary acts to the stage of automaticity of the low coordinating centers. As in vestibular nystagmus, some of these reflexes exhibit a series of to and fro movements, consisting of a principal movement

and then a movement of recovery, in preparation for a repetition of the principal movement.

7. Some direct evidence has been presented from experiments on animals, indicating that lower centers may initiate the quick component of vestibular nystagmus. Barenne, cited by Wilbrant and Sanger (*Neurologie des Auges*, achter Bond, *Die Bewegungssorungen der Augenmuskeln*, P. 308) observed no disturbance of nystagmus from turning in either direction, nor from caloric stimulations of either ear, in rabbits after removal of one cerebral hemisphere. Wilson and Pike in 1911 observed spontaneous labyrinthine nystagmus in a cat after destruction of the left labyrinth and the removal of the cerebral cortex of both sides. The optic thalami were intact in this experiment.

One of our experiments, (previously mentioned) although by no means conclusive, was not without significance. A cat under ether, was decerebrated to the extent that most of the cerebrum was separated from connection with the brain-stem. This was accomplished by an incision parallel and 1 c. m. anterior to the tentorium passing through the optic thalami. In spite of surgical shock from which the animal did not recover, a few movements of both slow and quick components of nystagmus were elicited by caloric stimulation of the labyrinth.

Resumé:

The quick component of vestibular nystagmus is initiated by the lower automatic mechanism, independently of the cerebrum. However, cerebral impulses can modify the action of the lower mechanism.

SPONTANEOUS VERTICAL NYSTAGMUS

Question:

Is spontaneous vertical nystagmus indicative of lesion within the brain-stem, or involving the brain-stem by adjacent pressure?

Against:

1. A small nystagmus in extreme position of lateral

gaze is recognized as physiologic. Why should we not also regard as physiologic a small vertical nystagmus, in the extreme positions, upward or downward, of vertical gaze?

2. Observations on normal individuals have demonstrated a small vertical nystagmus upwards, after prolonged fixation in the extreme upward direction.

3. In patients showing general neuro-muscular exhaustion, we have frequently observed prompt vertical nystagmus of fair amplitude, on extreme gaze, upward or downward.

4. Active vertical nystagmus, with the quick component upward, was observed by us in a cat upon recovery from ether, after injury of both internal ears. Necropsy showed that there was no intracranial injury.

For:

1. Patients showing general neuro-muscular exhaustion, without evidence of brain-stem lesion, have shown not only vertical nystagmus, but also horizontal, oblique and convergent nystagmus depending upon the direction of gaze.

2. Clinically, labyrinthitis produces a spontaneous horizontal nystagmus or rotary nystagmus, or, most frequently, a mixed horizontal and rotary nystagmus. We know of no case of proven end-organ lesion which has shown spontaneous vertical nystagmus, with the exception of the cat experiment noted above.

3. Brain-stem lesions frequently exhibit spontaneous vertical nystagmus.

4. Cerebellar lesions, angle tumors and other lesions in the posterior fossa, frequently show spontaneous vertical nystagmus. In our experience, vertical nystagmus has been observed in cases of posterior fossa lesion of the type of tumor or abscess—lesions producing local pressure. Others have made similar observations. However, that

these lesions produce nystagmus not intrinsically but because of pressure, has not been regarded as conclusive. A recent case, (Exhibit D.), is illuminating on this point. The case was one of large cerebellar abscess. Among other phenomena, there was a large spontaneous vertical nystagmus upward. The abscess was drained. After operation the vertical nystagmus was absent. Later, autopsy showed that the abscess was limited to the cerebellum. This case suggests that it was not the intrinsic cerebellar involvement that produced the nystagmus, but the pressure upon the brain-stem.

Resumé:

Spontaneous vertical nystagmus, per se, is not necessarily indicative of brain-stem lesion, for the reason that it has been observed as a fatigue symptom. However, spontaneous vertical nystagmus has frequently been observed in patients with lesions in or near the brain-stem and, so far as we know, has not been observed clinically in association with lesions in any other location. In patients who have other symptoms of intracranial lesion, a spontaneous vertical nystagmus indicates that the lesion is within or adjacent to the brain-stem.

PERVERTED NYSTAGMUS

Question:

Is perverted nystagmus indicative of lesion within the brain-stem or involving the brain-stem by adjacent pressure?

Definition:

We consider this phase of the subject of sufficient importance to describe the different types of abnormal eye responses to ear-stimulation, and to attempt a rough classification of perverted nystagmus.

1. Cases showing normal type of responses from stimulation of one ear, but abnormal responses from stimulation of the other ear.

2. Cases showing abnormal responses from stimulation of both ears.

3. Simple perverted nystagmus—in which the eyes are coordinated, but, the response is of a wrong type. Examples:—horizontal canal producing rotary, oblique or vertical nystagmus; or vertical canals producing horizontal, oblique or vertical nystagmus, instead of the normal rotary.

4. Inverse nystagmus, in which the nystagmus is diametrically opposite to the normal response.

5. Convergent nystagmus, in which each eye shows nystagmus inward.

6. Dissociated nystagmus, in which the nystagmus of one eye is of a different type from that of the other.

Against:

1. One should be guarded in pronouncing that a slight deviation from the normal type of nystagmus after stimulation is a true perverted nystagmus, because of the possible fallacy of imperfect technique. If one observes a rotary element in the response, after turning with the head 30 degrees forward, it would be wrong to draw hard and fast conclusions that this is a perverted nystagmus. Similarly, after douching with the head back 60 degrees, if one observe a rotary element, he must be sure to note whether the backward inclination of the head is at exactly the correct angle. It is also conceivable that the semicircular canals are not always in exactly the same plane in all individuals, as all physical structures occasionally show variation from the average normal. If, therefore on stimulation of the horizontal canal one observes a rotary element or a nystagmus in a slightly oblique plane, a certain latitude of interpretation is necessary. The abnormalities should not be considered as a true perverted nystagmus.

2. At each synaps in the course of nerve pathways, there is probably a regrouping of the functional relations

of the nerve fibers and a possibility of diversion of the pathways along which the impulse may pass. So long as an impulse is transversing one fiber, its course is fixed; at the synaps, there may be several avenues over each or all of which the impulse may continue. Every synaps in the course of the pathway increases the complexity of the pathways open to the impulse. Theoretically, if there is a block in any one segment of a pathway made up of several neurons, the impulse may deviate from its usual course by overcoming the threshold of collateral connections. One such a theory, Scarpa's ganglion might be the location where a lesion would divert the impulse from the horizontal canal to the pathways of the vertical canals. If this were true, it would be possible to account for a perverted nystagmus from a lesion not in the brain-stem, but within the internal auditory canal.

3. Even if the evidence were conclusive that perverted nystagmus is always of intracranial origin, indisputable evidence must be produced before we should state that it is indicative of brain-stem involvement. Supratentorial lesions, merely by increasing general intracranial pressure, may cause various localized phenomena, such as papilledema or abducens paralysis. It is conceivable that such general pressure might so derange the vestibulo-ocular mechanism as to produce perverted responses.

4. In the absence of conclusive data as to the exact mechanism involved in the production of perverted nystagmus, and in the absence of a great number of well-recorded and proven cases, it is unwise to regard this phenomenon as a positive indication of brain-stem involvement. To prove that it is not, all that would be required would be one case of frontal lobe tumor (or any other lesion at a distance from the brain-stem), which exhibited perverted nystagmus of any type.

For:

1. From our knowledge of the mechanics of the end-

organ and our interpretation of the normal physiologic responses, a perverted nystagmus is theoretically inconsistent with any disturbance of the end-organ or VIII Nerve.

2. The reflex pathway for normal nystagmus is from the vestibular end-organ, through the VIII Nerve and brain-stem to the ocular-motor nuclei. Theoretically it would appear that perversion of the normal response must be due to a disturbance along this pathway; if the end-organ and VIII Nerve can be excluded, this disturbance would appear to be necessarily within the brain-stem.

3. We know of no proven case of end-organ lesion which has shown perverted nystagmus. Neither do we know of any case of neuritis of the VIII Nerve which has exhibited this phenomenon. Tumors of the VIII Nerve frequently exhibit perverted nystagmus; here however, we have not only VIII Nerve lesion but a tumor mass distorting the brain-stem by pressure. It is a significant observation that in the tumors of the VIII Nerve that we have seen, perverted nystagmus never appeared as an early symptom but came on later, presumably in the course of the enlargement of the growth.

4. Lesions of the brain-stem itself frequently exhibit perverted nystagmus.

5. Lesions adjacent to the brain-stem of the type causing pressure, frequently show perverted nystagmus. It is sometimes noted, (as in case of Mrs. Fernandez, Exhibit E.), that perverted nystagmus comes on in the course of the development of a posterior fossa lesion; as the lesion progresses the nystagmus becomes perverted.

6. We have no record of any proven case of lesion not adjacent to brain-stem, which did exhibit perverted nystagmus.

Resumé:

Although this phenomenon has been observed for only a few years, and although the number of proven cases is

limited, yet it may be said that a true perverted nystagmus bids fair to be regarded as one of the most reliable signs of an involvement of the brain-stem.

• VESTIBULO-CEREBELLO-CEREBRAL TRACT

Before considering whether the impulses producing physiologic vestibular vertigo pass through the cerebellum, we should first consider the evidence from which we assume the existence of a pathway from the vestibular nuclei to the cerebrum. Everyone recognizes that the appreciation of movement is a concept that may be derived from various sensations, including visceral, tactile, muscle and vestibular sense. The full normal recognition of movement is unquestionably a composite of sensations. In this composite of sensations, the element that we are discussing is the vestibular sense. This, we regard as a specific special sense.

We observe that from each type of stimulation of the end-organ, affecting the internal ear in various planes and directions, there occurs, in consciousness, a specific sense of motion. This sensation is definite in regard to direction, plane, duration, intensity and rate of movement—always corresponding to the character of the stimulation employed. To our minds, this is proof that each impulse has reached the sensorium.

It is generally accepted that there is a cerebral center for each of the other senses; it would seem reasonable to postulate a similar cerebral representation for the reception of impulses from this end-organ of special sense. As to the location of such a center, there is little definite evidence. Certain evidence, however, has been presented by Dana, Mills and Cushing, suggesting that the temporal lobe may be the location of this center. Two cases of right temporal lobe lesions were reported by Dana; one had exhibited vertigo, and the other, vertigo and forced movements, backwards and to the right. Mills observed a case of tumor of the left mid-temporal region, in which vertigo was an early symptom and had persisted for five years. Cushing has noted the frequent presence of nystagmus, vertigo and incoordination in patients with temporal lobe lesions.

Whatever its location, this vestibular center is to be regarded

simply as the cerebral terminus for the reception of vestibular impulses—hence a *sensory center*. It is not an “equilibratory center.” Equilibration is a complex motor process. (Exhibit F., moving pictures of cats.)

Question:

Do the impulses from the internal ear, producing a sense of movement, pass through the cerebellum en route to the cerebrum?

Against:

1. So far as known, none of the other sensory pathways pass through the cerebellum. The visual and olfactory pathways enter the cerebrum above the brain-stem. Those of tactile, heat, cold, pain, pressure, kinesthetic, auditory and gustatory sensations, enter the cerebrum directly by way of the brain-stem. Lesions of the cerebellum are never characterized by any loss or any impairment of these sensations. If, as expressed above we consider that the impulses producing a sense of movement reach the cerebrum, from analogy of the other sensory pathways it would seem improbable that the vestibular impulses traverse the cerebellum.

2. Although fibres have been traced from the vestibular Nerve into the cerebellum, these may be accounted for as pathways concerned with functions not reaching consciousness. We have an analogy in the direct cerebellar tracts of the spinal cord, which enter the cerebellum via the inferior peduncles. according to the generally accepted view, these tracts, although they carry afferent impulses, are not concerned with sensations in consciousness. It would seem probable that the vestibulo-cerebellar fibres, like the direct cerebellar tracts, do not convey impulses concerned with the sense of movement, but constitute the afferent pathways for impulses to the cerebellum, resulting in coordinations of an automatic nature.

In consideration of these analogies, it might be inferred that the pathways conveying the impulses for the production of conscious sensations of motion from vestibular stimulation probably pass through the brain-stem, without any cerebellar connections.

For:

1. As before stated, fibres have been demonstrated to continue from the vestibular nerve into the cerebellum. Fibers have also been traced from the cerebellum into the cerebrum. These fibers traverse the superior cerebellar peduncles and decussate in the brachium conjunctivum. This arrangement would seem to furnish an anatomic basis for the theory that vestibular impulses pass through the cerebellum en route to the cerebrum.

2. Each case of proven cerebellar lesion that we have examined, has shown impaired or absent sensory responses from ear-stimulation. The experience of many others has corroborated this observation.

Resumé:

It would seem unquestioned that motion-sense impulses from the ear do reach the cerebral cortex. The location of the cortical center is not known; the only evidence that has been presented indicates its location in the temporal lobe. Although it is by no means proven, such evidence as is available suggests that these impulses traverse the cerebellum enroute to the cerebrum.

CEREBELLO-PONTILE ANGLE TUMOR PHENOMENON-COMPLEX

During the past six years we have observed the following phenomena in cases of angle tumor:

1. On the side of the tumor, a loss of auditory and vestibular function.
2. On the opposite side, normal auditory function, normal responses from the horizontal canal, but absence of responses from the vertical canals.

In brief, neuro-otologic examination showed a loss of function on both sides except the cochlea and horizontal canal of the ear opposite the tumor.

In some cases, during the early period of development of the tumor, we noted merely the auditory and vestibular loss of function on the same side. In the course of weeks or months,

one or more of the following additional phenomena were elicited:

1. The impairment and later, loss of responses from the vertical canals on the opposite side.

2. A spontaneous vertical nystagmus has been frequently observed. In certain cases, this vertical nystagmus was not observed on first examination but appeared after an interval. After such a case has once shown spontane-vertical nystagmus, we have never observed it to disappear, although we have noted variations in the amplitude of this verticular nystagmus.

3. In some cases, which had shown the complete phenomenon-complex, the horizontal canal on the opposite side had produced entirely normal nystagmus, vertigo and past-pointing. In other cases, during the early period of development of the tumor, the horizontal canal of the opposite side has shown normal nystagmus, but after an interval has produced perverted nystagmus.

4. In cases of well-advanced growth in the angle, we have observed abnormalities in past-pointing. This has been observed in a spontaneous past-pointing, and also in an absence or the impairment of past-pointing after ear-stimulation. In some cases, we have noted that we have termed a "crossed past-pointing;" both arms would past-point inward, or, both arms would past-point outward or, both arms would past-point outward, regardless of the type of stimulation. For example, the patient would past-point inward with both arms, regardless of whether he had been turned to the right or to the left.

It is difficult to interpret this phenomenon. In fact, our own experience has led us to draw no conclusions on the basis of abnormal past-pointing alone. Abnormalities in past-pointing have been observed in cases of cerebral lesion, as well as lesions within the posterior fossa; furthermore, we observed normal past-pointing in a patient who proved to have a cerebellar tumor. Although abnormalities in past-pointing do not appear, per se, to have anything approaching an exact significance or to furnish direct evidence in intracranial localization,

it would seem wise, however, to make careful tests of the past-pointing in all intracranial cases; abnormalities in past-pointing can at times be of distant value when correlated with other phenomena.

This phenomenon-complex has been observed and corroborated by many others in this country. However, we have no knowledge of its recognition in other countries. The only explanation of this phenomenon-complex would appear to be as follows:

1. The tumor destroys the function of the VIII Nerve on the same side by direct involvement of the VIII Nerve.

2. The isolated loss of responses from the vertical canals of the opposite side; the occurrence of spontaneous vertical nystagmus; the occurrence of perverted nystagmus on stimulation of the opposite horizontal canal; the abnormalities of past-pointing—all of these phenomena would appear to be caused by pressure of the tumor upon the pons.

Cautions:

Experience has demonstrated the need of caution in drawing conclusions in cases of suspected angle tumors. The number of cases of cerebello-pontile angle tumors, carefully studied by neuro-otologic tests, is insufficient to establish a complete working basis for interpretations.

We have recently studied a case, (Jesse Smith, Exhibit G.), which shows that other pathology than tumor can produce certain otologic phenomena of angle lesion. Jesse Smith showed involvement of the III, IV, V, VII and VIII Nerves, all on the right side. Ear tests showed complete loss of function in the right ear—both auditory and vestibular. Five days after the first examination, auditory function began to return; within two weeks, under our direct observation, instead of complete deafness in the right ear, there was such a complete restoration of hearing that he was able to hear a faint whisper, with the noise apparatus in the left ear. The caloric test also demonstrated the return of vestibular function. The vestibular function also returned within a period of two weeks. The facial paralysis also disappeared. The neurologic diagnosis was mul-

multiple sclerosis. In this case it is to be noted, however, that there was no impairment of responses from the vertical canals of the opposite side.

In the earlier stages of angle tumors, the phenomenon-complex is incomplete. Most assuredly, a diagnosis of angle tumor is not to be made, based only upon abnormal or absent responses from the one ear. Given, however, a picture of absent auditory and vestibular function in one ear; impairment or perversion of responses from the opposite ear, with normal auditory function in the opposite ear—we have data indicating involvement of the angle.

Even under these circumstances, however, one cannot state that there exists a tumor which is confined to the VIII Nerve and is probably removable. We have seen this phenomenon-complex in tumor of the pons and tumor of the lateral cerebellar hemisphere, invading the angle.

It is occasionally possible to draw valuable inferences from the history of such a case. If, over a considerable period, all the symptoms have been of the type referable to VIII Nerve involvement, and then there begin to appear the absence of vertical canals responses on the opposite side and other phenomena of brain-stem involvement, we are justified in stating that the evidence makes probable a true neuroma of the VIII Nerve. (Miss Buchart, Exhibit H).

The first symptoms of angle tumor are frequently those referable to functions of the VIII Nerve; often it is the otologist who is first consulted by patients with this lesion. While the diagnosis of angle tumor is not dependent upon neuro-otologic information, this phenomenon-complex has been observed so frequently in proven cases that its presence may now be regarded as a valuable diagnostic sign.

NEURAXIAL DIFFERENTIATION OF THE FIBRES FROM THE HORIZONTAL CANAL AND THE FIBRES FROM THE VERTICAL CANALS.

Before discussing this problem, it is necessary to state exactly what is meant by this neuraxial differentiation. Our con-

ception of this differentiation is that there is a divergence of vestibular fibres within the brain-stem, in such a manner that the fibres in relation to the vertical canals are separated by appreciable distance from those in relation to the horizontal canal.

This separation is considered to be sufficient to permit macroscopic lesions to exert a selective effect upon one set of fibres and not upon the other.

Here it should be noted that the various vestibular responses have generally been described in terms of the semicircular canals, without recognition of the probable cooperation of the otolith organs of the utricle and the saccule in the production of these responses. The work of Maxwell has made clear that the utricle is a definite factor in the production of reflex ocular movements; we should always bear in mind the probability of the participation of the otoliths of the utricle and saccule, to a greater or less extent, in the registration of movement in any plane. Therefore, although we are unable to estimate the exact relationship of the different nerve receptors, and consequently, for convenience, speak in terms of the semicircular canals, yet we should not lose sight of the part that may be played by the otolith organs.

Question:

After entering the brain stem, do the fibres from the horizontal canal pursue a different course from those conveying impulses from the vertical canals?

Against:

1. No histologic proof of these separate pathways has been furnished. There are many vestibular pathways which have been traced histologically:

(a) Fibers from the vestibular portion of the VIII Nerve have been traced to the so-called vestibular nuclei in the brain-stem—the Deiters', Bechertow and Triangular Nuclei. These nuclei occupy an area of considerable size and extent throughout the lower portion of the pons and the upper portion of the medulla oblongata.

(b) Fibers from the vestibular portion of the VIII Nerve have been traced into the cerebellum without interruption.

(c) Fibers from the vestibular nuclei, by association and commissural pathways, have been traced in various directions, to the posterior longitudinal bundles, into the spinal cord and the corpora quadrigemina, of both the same and opposite sides.

All that is known of these nuclei and fiber tracts within the brain-stem and cerebellum, is that they bear some relation to the vestibular portion of the VIII Nerve. No nucleus or separate fiber tract has been identified as being in relation with the utricle or saccule or any semicircular canal.

2. Functional differentiation of fibers does not necessarily imply a divergence of their anatomic course. Pathologic processes may involve certain fibers, while others in close proximity escape. This may occur even within a nerve trunk, as in degeneration of the papillo-muscular bundle of the optic nerve. The destruction of this particular bundle of fibers within the optic nerve has been histologically demonstrated in patients who have shown blindness of the central portion of the visual field. A similar selective process may occur within the VIII Nerve; it is not a rare observation that in a neuritis of the VIII Nerve, as in syphilis, the cochlear portion may be affected and the vestibular portion not affected, and vice versa. It requires no stress of the imagination to conceive that within the VIII Nerve there may be an involvement of the particular fibers in relation to one semicircular canal, without the involvement of the rest of the nerve. Similarly, at any point throughout their intracranial course, it would seem that a pathologic process might affect certain fibers without affecting those even immediately adjacent.

3. The only evidence in favor of separate pathways from the different canals has been the clinico-pathologic. In the experience of all who have made vestibular tests of intracranial cases, it has been the vertical canals that have

shown impairment far more than the horizontal canals. The non-response of the vertical canals should not be regarded as conclusive evidence that the vertical canals fibers are widely separated from the horizontal canal fibers. It would seem reasonable to consider that the vertical canals fibers may be less resistant; the horizontal canal mechanism is the more active, the more frequently used, and unquestionably the more primitive. It would seem that the horizontal canal mechanism might be more resistant, because of its being more firmly established in function than that of the vertical canals.

For:

1. The conception of separate neuraxial pathways from the different canals was advanced in 1916, on the following data:

In a series of cases with intracranial lesions, in which the labyrinth and VIII Nerves were normal, the vertical canals failed to produce responses.

These cases included lesions of the pons, angle tumors pressing against the pons, cerebellar lesions causing pressure against the pons, absence of the IV Ventricle and internal hydrocephalus with pressure within the IV Ventricle.

These cases suggested the following:

If the lesions involving the pons caused a block in the responses from the vertical canals without impairing the responses from the horizontal canal, and as in all these cases the medulla oblongata was uninvolved, it was considered probable that the fibers from the vertical canals ascended into the pons, probably close to the floor of the IV Ventricle, whereas the fibers from the horizontal canal presumably traversed the medulla oblongata.

Confirming this viewpoint were the findings in the case of thrombosis of the posterior inferior cerebellar artery; all responses from all canals were normal, except that the right horizontal canal failed to produce vertigo and past-pointing.

This lesion, thus involving a portion of the horizontal

canal pathways, is known to involve the right side of the medulla oblongata in the region of the right inferior cerebellar peduncle.

2. Much confirmatory evidence of the same character as the above has accumulated. Eagleton had previously noted that in cases of angle tumors, the caloric test from the opposite ear gave negative responses; later, Eagleton and others, observed that in such cases, when the head was tilted backwards sixty degrees stimulating the horizontal canal, the responses came through.

It is now a common observation that there is a non-response of the vertical canals in lesions of the posterior fossa involving the brain-stem by infiltration or pressure. Few cases, however, have been seen in which the horizontal canal showed no response, while the vertical canals show normal responses.

3. We have recently seen two cases, which, however, have not been confirmed by necropsy, in each of which the left horizontal canal failed to produce responses, whereas the responses from all of the other semicircular canals were normal.

4. Although no histologic demonstration has been made of separate pathways from the different nerve receptors in the end-organ, yet there is evidence that the vestibular nerve fibers spread out to be distributed to the vestibular nuclei. The fibers from the vestibular portion of the VIII Nerve are for the most part interrupted in these nuclei, within a short distance from their entry into the brain-stem. They enter the brain-stem as a compact bundle, but their nuclear connections require a physical separation of all the fibers, preliminary to their termination. Complete ultimate physical dissociation of all these fibers must be accomplished, since each fiber has its definite destination in the vestibular nuclei, different from all others. These nuclei cover a large area; they involve the lower portions of the pons and the upper portion of the medulla oblongata and

extend from the lateral aspect of the brain-stem well over toward the median line. Without speculation as to the ultimate course or destination of the neurons from the various portions of these extensive vestibular nuclei, the physical separation of cell-groups in these nuclei—regarded as representing end-organ units—would in itself constitute a neuraxial differentiation of sufficient degree to answer the requirements of the concept given in the question.

Resumé:

While much remains to be learned of the intramedullary pathways and connections of the vestibular fibers, the evidence indicates that there is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal and of the fibers in relation to the vertical canals.

V. SUMMARY

Theoretical:

1. The available evidence suggests that the cortical area for the reception of impulses from the vestibular end-organ, is located in the temporal lobe in each hemisphere.

2. Different cell-groups within the vestibular nuclei represent the different physical units of the end-organ.

It is Probable:

1. That the otoliths and the cristae of the semicircular canals cooperate in function.

2. That the vestibular fibers are not distributed to the peripheral motor neurons, but to the neurons of the ocular coordinating mechanism.

3. That the quick component of vestibular nystagmus can be produced by a lower mechanism, without impulses from the cerebrum.

4. That the impulses from the internal ear, producing a sense of movement, pass through the cerebellum enroute to the cerebrum.

REASONABLY PROVEN: *To be relied on in clinical work.*

1. There is a cerebral cortical center for hearing.
2. This center is reasonably proven to be in the temporal lobe. Also that each ear is in relation to the temporal lobe of each hemisphere.
3. Capillarity does not prevent endolymph movement within the semicircular canals.
4. There is a coordinating mechanism for ocular movement within the brain-stem.
5. Spontaneous vertical nystagmus, in patients who have other symptoms of intracranial lesion, indicates that the lesion is located within or adjacent to the brain-stem.
6. Perverted nystagmus after ear stimulation is indicative of involvement of the brain-stem.
7. Motion-sensing impulses from the end-organ, are conveyed to the cerebral cortex.
8. Tumors of the cerebello-pontile angle frequently show a typical neuro-otologic phenomenon-complex.
9. There is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal and of the fibers in relation to the vertical canals.

THE STUDY OF THE TONAL RANGES IN LESIONS OF THE MIDDLE EAR.

By L. W. DEAN, M. D. and C. C. BUNCH, Ph. D., Iowa City, Ia.

The research work which we are reporting represents a large amount of careful, conscientious work especially on the part of Mr. Bunch, who either made or supervised all the functional tests of hearing which are indicated. This report is confined to the examination of the auditory perception of a large series of cases of so-called middle ear trouble. In all cases exhaustive clinical examination was made. These examinations were grouped and studied and the results are here summarized and presented for your consideration.

PLATE 1 is the diagram of the clinical record made for each case examined. In only a few cases where it seemed desirable to have specific information regarding the vestibular functions were the turning tests made.

The hearing for whispered and spoken voice was first determined. Next the pitch range audiometer was used to determine the audition for tones between 30 and 7070 d. v.

The records with the audiometer are easily indicated by means of a graph shown in the plate. The pitch of the tones is indicated at the bottom and the loudness of the tone by the height of the curve on the graph, no tones at all being normally heard at zero. The curve shown on the graph is the field of hearing for a normal ear.

In the curves of cases studied that we are going to show, the curve was secured by testing the audition of the ear examined for every tone from 30 d. v. to 7070 d. v. The curve thus secured compared with the normal curve which is stamped on every chart gives an approximate idea of the relative hearing power for these tones.

If, for instance, the curve at 1,000 d. v. is only half as high as normal, this does not mean that hearing for that note is one-half normal but only that hearing for the note is markedly decreased. We are not justified in assuming that the curves represent the exact audition for the note that is being tested. The fractions which express quantitatively the audition for various notes, when this is determined with the Bezold forks, are likewise very inexact. As the intensity of sound varies with the square of the distance, with the square of the amplitude of the vibration, and is influenced by the rapidity of vibration of sound it is an extremely difficult matter to determine in any way the exact amount of audition for any given note.

If the curve of the ear being examined should show a tone gap in the tonal range, this does not necessarily mean that there is an absolute tone gap but it does indicate that the ear is unable to perceive the loudest tones capable of being produced by the audiometer. These gaps found by the audiometer have been verified in the psychological laboratories by tests with organ pipes and other instruments, producing pure loud tones. It is easy to conceive that if a note could be produced of greater intensity than that which the audiometer can give it might be heard when the audiometer would produce no sensation of sound.

In the following charts the normal curve is indicated by a continuous line; curves for the right ear by dashes; and for the left ear by dots.

For the benefit of those who are not interested in the audiometer readings we have made in each case a quantitative determination of the audition for the 50 d. v. fork, the c , c^1 , c^2 , c^3 , c^4 , and c^5 forks.

The Rinne, the Weber, and the bone conduction tests were made with the 256 fork. In some cases the 128 and 512 d. v. fork was also used. These forks were weighted to minimize the production of confusing overtones.

At the bottom of the plate next to the audiometer curve is a measurement of the upper tonal limit as determined with the Galton whistle, the Koenig cylinders, and the

Monochord. The upper limit of the monochord is determined both by A. C. and by B. C. With the Galton whistle which we use the normal upper limit is 21,000 d. v. With the Koenig cylinders the upper limit is 43,960 single vibrations and with the Monochord by A. C. 19,000 d. v., by B. C. 20,000 d. v.

In each case studied we have been interested in determining if in addition to the middle ear lesion there was also present a lesion of nerve or end organ.

Suggestive of a lesion of the acoustic nerve or its end organ is the following:

1. The audition for the higher notes is poorer than that for the lower or middle part of the tonal range. If the lower notes of the tonal range up to about 3,000 d. v. are heard normally and there is a diminution in the audition for the notes in the upper part of the range as is shown by PLATE 2, this diminution of audition for the higher notes is suggestive of a lesion of the nerve or end-organ. If, however, as is shown in PLATE 3, there is a uniform diminution in audition for tones throughout the range it does not indicate a lesion of nerve or end-organ. The curves shown in PLATE 3 are the curves secured from; first, a case of impacted wax in the external canal; second, a similar case; third, three cases of individuals with normal hearing where the external canal has been packed with borac acid powder, and lastly, a case of cotton in the ear which was soaked with cerumen and was in contact with the drumhead. In each of these cases you note the diminution in audition for all tones throughout the range. As we view it, there would have to be a distinctly greater diminution for the notes in the upper part of the range as compared with the tone best heard in the lower or middle part in order to indicate positively a lesion of the nerve or end organ. In some of these cases the c^5 fork was not heard at all.

PLATE 4 shows the curves of these cases after the removal of the impacted wax, cotton or borac acid powder. Note that the curves are practically normal.

2. A lowering of the upper tonal limit, especially by B. C. The better the hearing for voice and whisper, the more significant of a lesion of nerve or end organ is the decrease in the upper limit by A. C.

3. Positive Rinne.

PLATE 5 indicates the upper tonal limit in three of the cases of obstruction in the external canal just described by A. C. and B. C. determined with the monochord. Note the upper limit by A. C. is lowered 2,000 d. v. from obstruction in the external canal. There is, however, no cutting down of the upper limit by B. C. and we feel that a lowering of the upper limit by bone conduction is more definite indication of lesion of nerve or end organ than the lowering of the upper limit by A. C.

PLATE 6. The comparison of the upper limit of audition for high notes as determined by A. C. and B. C. is of interest.

The upper chart shows the A. C. and B. C. as determined with the monochord for twenty-two acute suppurative ears. The vertical figures represent the number of cases; the horizontal the upper limit in double vibrations per second. The curves are similar but by air the lowest limit was 12,000 d. v. while by B. C. it was 14,000.

The results of examination of twenty-eight cases of chronic otorrhea are chartered in the center. Again the larger number of cases make similar curves but here, while the lowest limit by B. C. is 13,000 d. v., by A. C. it is 6,000.—a great difference.

Below are graphs secured for charting the results of forty cases of hyperplastic otitis media. Here the similarity of the curves is lost.

The diagnostic significance of the difference between the upper limit as determined by bone and air conduction with the monochord is here illustrated. In cases of acute trouble

22% gave a difference of 3,000 d. v. or more between the upper limits determined by air conduction and by bone conduction. In the chronic otorrhea cases this percentage was 39 while cases of hyperplastic otitis media gave this difference in 67%.

The determination of the upper limit by B. C. is a far more accurate indication of the condition of the nerve and end organ than its determination by A. C. .

4. The sensitivity for one note or a group of notes is much less acute than the others when determined with the **tuning fork or the audiometer.**

To determine whether there is a significant difference between the hearing power for high tones and low tones, we have used the audiometer and the Bezold forks. For example, if the hearing for the c^2 fork is indicated by the fraction 20/24 while that for the c^5 fork is indicated by the fraction 5/60, there is a greater decrease for the c^5 than for the c^2 . This would be shown on the audiometer record by a curve which closely follows the normal curve for a frequency of 512 d. v. but which falls much below the normal curve at 4096 d. v.

PLATE 7 is a plate of neuro-labyrinthitis showing these distinct depressions in the audiometer curve confirmed by the findings with the tuning forks.

5. Decreased bone conduction as determined by the c^1 and c^2 fork.

This is a very important matter. We feel that a bone conduction decreased two or three seconds is suggestive of a lesion of nerve or end organ when the examination had been properly made. We test bone conduction by placing the tuning fork over the bone just adjacent to the external opening of the bony canal posterior to the upper attachment of the external ear. We keep in mind the influence of mastoid involvement decreasing the bone conduction.

Bezold (Text Book of Otology, page 67) thinks that to be significant the perception by bone conduction for the fork placed on the vortex should be very much shortened.

Wittmaack (*Zeit. f. Ohr.* vol. 60, page 136) is of the opinion that a shortening of the bone conduction is worthy of diagnosis when it amounts to at least 30% of the average value which we get or receive through the examination of a large number of normally hearing individuals with the chosen tuning fork.

As the percentage of decrease will depend altogether upon the energy with which the fork is excited, this statement is of little value.

Bezold feels that an objection to this procedure is that extraneous sounds and the ability of the individual to make an accurate examination and of the patient to cooperate have a decided bearing upon the result. Such criticism is valid for all functional tests of hearing and is as true for determining hearing power by A. C. as well as B. C.

Our examinations are conducted in a room set apart especially for this purpose and, while it cannot be said to be entirely free from external noises, it is probably much more nearly ideal for this purpose than those ordinarily used.

Emerson (*Annals of O. R. & L.* Dec. 21, page 999), is of the opinion that when B. C. is lowered for the 256 or 512 d. v. fork that nerve deafness has been present for a long time.

Werhofsky (*Prufung, d. Hordauer in Verlaufe der Ton-skala bei Erk. d. Mit. u. Innern O.* Bezold U. d. Funk, *Prof. d. Mench. Gehor*, 1897-1909) found decreased B. C. in all lesions of the nerve or end organ that he studied. He reports B. C. decreased two seconds in one case of nerve deafness and three seconds in another.

He says that B. C. is less with higher forks than with lower and cites a number of observations to confirm this.

In describing his technique he does not state the length of time his forks vibrate with a given stimulus. Our higher forks vibrate longer than the lower and consequently the decrease in B. C. with the higher forks would be greater.

Siebenmann in an article translated by Dench (*Archives of Otology*, vol. 22, Pg. 5) says that in apparently normal ears there may be functional defects in the test for the

upper tone limit and in the perception of sound by B. C. He also shows that B. C. is sometimes decreased by aspiration of the middle ear.

Page, (Proceedings N. Y. Academy of Med. Oct. 13, 1921) says that the early cases of otitis media have a negative pressure in the tympanum.

Kerrison (Text Book of Otology, Pg. 89) suggests that the hearing by bone should be decidedly decreased to be significant.

Schwabach (Über Hörprüfung, etc. Archiv. f. Ohren. vol. 31, page 6) says that B. C. is longer than with normal persons in lesions of the transmitting apparatus while it is the same as normal persons or less in lesions of the perceiving apparatus of the ear.

In our study of acute middle ear infections, we have found it very unusual to find an infected ear without evidence of a lesion of its nerve or end organ.

Siebenmann, in an article translated by Dench (Archives of Otology, Vol. 22, Page 12) reports that in making functional examinations of pure cases of catarrh of the eustachian tubes, he found the upper tone limit usually lowered and not restored by inflation. He assumed that the lowering of the upper limit was due to a passive hyperemia of that portion of the scala vestibuli lying next the oval window.

PLATE 8 shows curves of a case of acute tubal catarrh where we found no definite evidence of lesion of nerve or end organ. The decrease in hearing for the voice and whisper was apparently due wholly to the middle ear condition and disappeared on inflation. B. C. in each ear was normal. Upper limit by Monochord, R., A. C. 16,000, B. C. 19,000; L., A. C. 17,000, B. C. 19,000. As with the blocking of the external canal the upper limit was lowered by air and not by B. C. Unfortunately, after inflation the upper limit was not tested by A. C.

The curve before inflation shows rather a uniform decrease in audition for all notes except the very lowest which

were not heard at all. The audition for the notes became almost normal on inflation.

Twenty-two acute infected ears were studied. Of the twenty-two, fourteen had positive Rinne and eight had negative Rinne; fifteen had decreased bone conduction for the c^2 fork and fourteen had the upper tone limit decreased 2,000 or more by A. C. as determined by the monochord. Thirteen of the fourteen had the upper limit by B. C. decreased 2,000 or more. Twelve cases had decreased bone conduction as well as lowered upper limit. Two cases had decreased B. C. with upper limit normal and two cases had upper limit by A. C. lowered but B. C. normal.

PLATE 9 shows the curves of ten acute infected ears. The dots, dashes and circles in the curves are added so that they may be more easily followed through the entire field. You will note that curves No. 2 and 3 show complete gaps. Partial gaps are shown in all the curves except No. 4. 90% of the curves of acute infected ears are suggestive in this manner of lesions of the nerve or end organ.

A careful analysis of the clinical charts of the twenty-two ears studied shows that in every acute infected ear except one there was either decreased B. C., upper limit lowered by B. C. 2,000 or more, or a defect in the audiometer curve suggestive of lesion of nerve or end organ.

These evidences of lesion of nerve or end organ appear very early in the course of the disease.

PLATE 10. H. B. Examination was made within twelve hours after first evidence of infection. With good hearing for voice, spoken, 45 feet, and whisper, 15 feet, in each ear; frank earache in the left, slight earache in the right; drum-heads reddened, no bulging; Rinne positive in each ear, c^5 fork in left 30/60; with other forks, normal. Two partial tone gaps in left ear with audiometer curve. B. C. -2 sec. each; upper limit lowered 2,000 by B. C. with monochord. No leucocytosis at this time. Patient discharged ten days later, B. C. normal. c^5 heard normally,—upper limit normal by A. C. and B. C.

In cases where the infection was unilateral the evidences of a lesion of nerve or end organ were not confined to the infected ear.

PLATE 11 shows the findings of the good ear in six cases of unilateral acute infection of the middle ear. Only one case, the first, had no suggestion lesion of end organ or nerve in the good ear. In the second, third, fourth and fifth cases the upper limit is lowered; in the fourth, fifth and sixth B. C. is decreased. The audiometer curves of the first case, and of cases three and four, were somewhat suggestive of lesion of nerve or end organ so that in at least five out of six of the unaffected ears and probably all six, there is evidence of involvement of the nerve or end organ.

PLATE 12 B. D. This case is interesting because a few days before infection of the left ear the ears were tested and were apparently normal. Note that in the right ear where there was no redness of drumhead or other evidence of middle ear infection, the decreased hearing for voice and whisper, the decreased B. C., the decreased upper limit by B. C., and the slight diminution of audition for the c^1 , c^2 , c^3 , c^4 , and c^5 forks, for the left ear, the decreased B. C., the tuning fork tests and the audiometer curve are all suggestive of lesion of nerve or end organ.

Treatment and eradication of the middle ear condition may have no influence upon the lesion of nerve or end organ.

PLATE 13 E. B. shows the findings at the height of an acute tubal catarrh. The tubal orifices were edematous covered with dilated blood vessels; the drumheads were reddened, not bulging. B. C. decreased each 2 sec. Upper limit each B. C. 17,000 by monochord.

PLATE 14 shows the findings after the eradication of the middle ear lesion. B. C. -3. Upper limit lowered by B. C. Audiometer curve indicative of lesion of nerve or end organ. The findings remained the same as long as the patient remained under observation.

PLATE 15 shows the findings A. A. S. Feb. 27, taken one month after an infection of the left ear was apparently well. Curve and tuning fork findings for left ear indicate a lesion of importance involving nerve and end organ. Whisper and voice are poorly heard.

PLATE 16 B. D. Sept 27, shows findings in both ears in a case of acute infection of the left ear one month after discharged as cured. The curve left, tuning fork tests left, and decreased B. C. each indicates a lesion of nerve or end organ left and probably also right. At no time during the disease was there evidence of acute middle ear infection right. Nevertheless, the hearing right which was W15, V45, before the infection of the left ear is now W13, S45, while the B. C. is -5. Before the infection of the left ear it was normal.

After acute infections of the middle ear with return to normal audition for voice and whisper there may be definite indications of lesions of nerve or end organ.

Twelve cases of bilateral chronic otorrhea and fifteen cases of unilateral chronic otorrhea, or thirty-nine ears, suffering with chronic otorrhea, were studied. Rinne was negative in thirty-five ears, positive in four. There was decreased bone conduction in eleven ears, increased in twenty-six, normal in two. Decreased B. C. was not so common as in the acute infections. The upper tone limit was decreased 2,000 or more by bone conduction in thirty ears. In thirty-seven ears the audiometer curve was suggestive of a lesion of nerve or end organ and in two ears not. Marked depressions and tone gaps in the audiometer curves were much more frequent than in the acute ears. The curves were much more characteristic of lesions of the nerve or end organ. If we include the results with the tuning fork tests in only one ear out of the thirty-nine, was there no evidence of a lesion of the nerve or end organ.

PLATE 17 R. B. is a typical chart showing the findings in a case of chronic otorrhea. Note that with the tuning forks the hearing range is from c^1 to c^4 , inclusive. The

audiometer curve shows the decreased audition for both low and high notes in the range of the audiometer with depressions characteristic of nerve or end organ. The upper limit in each ear is decreased to 15,000 d. v. by B. C.

From a study of the cases of chronic otorrhea one gets the impression that the extent of the lesion of nerve or end organ corresponded roughly with that of the middle ear lesion, while in the acute cases there was the greatest variation between the extent of the lesions of the inner ear and those of the middle ear. It was not unusual with a very mild acute middle ear infection to have a very well marked lesion of the nerve or end organ. A study of the fifteen unaffected ears in the fifteen cases of unilateral chronic otorrhea shows that Rinne was negative in unaffected ears no times, positive in fifteen. B. C. was decreased in the unaffected ears three times; it was normal ten times and increased twice; the upper limit was decreased by B. C. 2,000 or more five times and was normal ten times. The audiometer curve of the unaffected ears was indicative of a lesion of nerve or end organ in only one case. The unaffected ears in this series of unilateral cases were very good ears with one exception. The hearing in almost every ear was W15 feet. While these findings suggest that there are a fair number of lesions of the nerve or end organ in the unaffected ears in chronic otorrhea we do not find the large percentage of involvement of the nerve or end organ as in the unaffected ears in the acute infections. This suggests to us that the involvement of the nerve or end organ in the unaffected ears in acute infections is frequently not a permanent affair; otherwise, we would find the same number of lesions in unaffected ears in chronic otorrhea. The lesions of the nerve or end organ in ears suffering from chronic otorrhea were much more marked than in the acute infections. There is a direct interdependence between the chronic otorrhea and the lesions of the nerve or end organ. The chronic otorrhea has no influence of any moment upon the nerve or end organ of the opposite side. In our acute cases we get the impression that the acute middle ear

trouble and the lesion of the nerve and end organ are not interdependent. They may both be due to the same cause but not necessarily so.

PLATE 18 is a composite graph of the results obtained from tuning fork examinations. It is difficult to express in a uniform manner the results obtained in tuning fork examinations when different denominators are used to represent normal hearing for different tuning forks. For example; in measuring the hearing power for the tuning forks we represent the hearing for a normal ear in terms of distance as the denominator of a fraction. The hearing power for that fork by a pathological ear is recorded in the same terms in the numerator. For the c^5 fork, since a very slight impact produces a sound heard at a considerable distance, we use the denominator 60. The 256 fork is heard at a less distance and we use 24 for the denominator. For similar reasons we use the denominator 4 with the 50 d. v. fork.

In order to make an analysis of our results we have arbitrarily divided our results into six classes; those who do not hear the fork at all, are put in class 1, those who hear the fork when it is very loud as indicated by fractions equivalent to one-fourth or less we have put in Class 2; those who hear it by values indicated by the fractions between one-fourth and one-half are put in Class 3; those who hear it by fractions from one-half to three-fourths are put in Class 4; those who hear it from three-fourths to less than normal are put in Class 5; and those who hear it normally are put in Class 6. The curve shows that twenty-one cases did not hear the 50 d. v. fork at all; three did not hear the c^5 fork; fourteen heard the 50 d. v. fork with a fraction equivalent to one-fourth or less; while twenty-seven heard the c^5 fork with results indicated by a fraction of one-fourth or less. You will notice that the 512 d. v., 1024 d. v. and 2048 d. v. forks run almost parallel throughout the entire curve while the greatest variations in chronic otorrhea cases are shown with the 50 d. v. fork and the 4096 d. v.

fork. There were no cases where the hearing was perfect for the low fork and two cases where the hearing was normal for the 4,096 d. v. fork.

PLATE 19 shows the results of the audiometer examinations in ten cases of chronic otorrhea. It is inserted to show the frequency with which such phenomena as tone islands, gaps and other peculiarities exist. These peculiarities are indicative, in our judgement, of lesions of the nerve or end organ. For example, curve No. 8 shows simply a region of residual hearing for tones between 500 d. v. and 1,500 d. v. No. 7 while rising much higher on the chart, shows two islands with the interlying gaps at 2,500 d. v. and at about 3,200 d. v., Curve No. 9, even though rising to the normal at 1,000 d. v., shows that the low tones are lost, that there is a decrease for notes above 1,200 d. v. and a return to almost normal at 5,500 d. v.

Sixty-six percent. of the records of cases with chronic otorrhea show either complete or marked partial tone gaps. These do not include such cases as are illustrated by curves No. 1, 2, and 8 which are equally significant of an inner ear lesion.

In only one case L. P. age 13 did a tone gap disappear with treatment.

Hyperplastic Otitis Media. Thirty cases of bilateral H. O. M. and four cases of unilateral H. O. M., or sixty-four ears were studied. Rinne was negative in thirty-three ears, positive in thirty ears, questionable in one ear. There was decreased bone conduction in forty-four ears, increased bone conduction in sixteen ears, normal in four ears. The upper limit was decreased by B. C. 2,000 or more d. v. in forty-nine ears.

Twenty-one ears had complete tone gaps in the audiometer findings. In eight more there were marked partial gaps or 45% showing these peculiarities. In addition to this there were 34 cases where there were marked depressions equally indicative of inner ear involvement.

PLATE 20 shows the curves of ten typical cases of H. O. M. Complete tone gaps are illustrated in curve No. 10

which has a gap at 2,400 d. v. and another at 2,000. Curve No. 9 shows a dip or partial gap at 1,600 d. v. No. 6, while not showing either a partial or complete gap shows that the hearing was normal up to 1,500 d. v. and decreased more and more for tones higher in the scale. A curve of this type certainly indicates a lesion of the nerve or end organ. Curve No. 8 shows only a patch of residual hearing between 100 d. v. and 1,500 d. v. Each curve shows marked diminution in the audition for the c^5 fork. In the next plate you will note that in only eight of the sixty-four ears was the c^5 fork heard normally.

PLATE 21 shows a composite of the tuning fork cases in hyperplastic otitis media. Here we have made the same arbitrary divisions as we did with Plate No. 18. Here again we see that the larger variation is shown with the 4096 d. v. and the 50 d. v. forks. The 1024 d. v. and the 2048 d. v. forks run almost parallel through the entire range. While there were no cases where the 4096 d. v. fork was not heard there were 51 cases who could hear this fork only when it was struck very loud as, for instance, when the fork was forcibly struck against the heel of the shoe, twenty cases did not hear the 50 d. v. fork.

Note that in eight cases the 50 d. v. fork was heard apparently normal. In these eight cases the lesion of the nerve or end organ over-shadowed the middle ear lesion.

A study of the clinical charts of these cases shows evidence of the involvement of the nerve or end organ in every ear affected save one.

PLATE 22 F. K. shows the curves of a bilateral case of H. O. M. Note the negative Rinne, slightly decreased B. C., power of hearing low and high notes markedly interfered with in each ear, tone gaps in the audiometer curve, and upper limit markedly decreased both by A. C. and B. C.

PLATE 23 C. L. shows in the right ear bone conduction normal, and negative Rinne, audiometer curve practically normal, upper limit normal. In the left ear B. C. is decreased five seconds. The right ear of this case was the

only one of the sixty-four ears studied that did not show some evidence of involvement of nerve or end organ. Hearing was poorer in the right ear.

There was found no definite relation between the extent of the inner ear lesion as compared with the middle ear lesion.

PLATE 24 shows the findings in H. S. aged 62, and in C. P. aged 62. The objective findings of the middle ear were similar. In one case the bone conduction is -5 seconds and in the second case it is -12 seconds. In the first case, the audiometer curve shows a tone gap in the left ear and in the second case there is none. In the audiometer curve the upper notes are heard much poorer in the first than in the second. In the first case the upper limit is so low that it cannot be detected using the Galton whistles or the monochord either by A. C. or B. C. and with the Koenig cylinders it is only approximately half normal 21,000 s. v. instead of 41,000, while in the second case the upper limit is decreased, it is 16,000 by B. C. with the monochord. The lesion of the nerve or end organ is much more marked in the first than the second case. In the second case, the one with the least involvement of the nerve, had a positive Wassermann, the first one not.

PLATE 25 (Findings of Miss D. and H. B.)

The age of the first is twenty-five years and the age of the second is twenty years. The hearing in the left ear is approximately the same for whisper. In the first case B. C. left is increased, the tuning fork test shows fair hearing for the c^4 and c^5 fork, the upper limit by B. C. is normal, the audiometer curves, while there is a gap in the right ear and depressions in the left shows fair hearing for some of the notes in the higher part of the range. In the second case, however, B. C. for c^1 fork is -5, c^2 fork -6, hearing for the c^4 and c^5 fork is markedly decreased, the audiometer curves show a cutting down of the higher notes to 4,000, the upper limit by B. C. is 15,000. The evidence points to a much more marked lesion of the nerve or end organ in the second case than in the first.

In one case of H. O. M. while under observation acute suppurative otitis developed. With the diminution in hearing the B. C. which was plus 4 in the left ear became decreased. With the disappearance of the acute otitis it became plus again.

Our cases of H. O. M. frequently tell of improvement in hearing that is satisfactory to them following one kind of treatment or another. Our tests have failed to show any actual improvement in any case unless the lesion of the nerve or end organ greatly over-shadowed the middle ear lesion and the improvement resulted from the treatment of the case for syphilis, or removal of tonsils, or whatnot.

PLATE 26. H. J. is a case of H. O. M. where there was also a plus 4 Wassermann. Note the positive Rinne with the c^2 fork and the greatly decreased B. C. with the same fork and a negative Rinne with the c^1 fork which is decreased only six seconds. This patient's deafness has rapidly appeared in the last six months. It has progressed too rapidly for H. O. M. The syphilitic lesion of the nerve or end organ is more responsible for the deafness.

PLATE 27. J. C. is a case of H. O. M. with negative Wassermann and no evidence of syphilis where the lesion of nerve or end organ greatly over-shadowed the middle ear lesion. Note the Rinne plus, B. C. decreased 6 seconds and 4 seconds, the tuning forks suggestive of lesion of nerve or end organ. The upper limits by B. C. 12,000 d. v. and 13,000 d. v. The curve is also characteristic of lesion of nerve or end organ.

A study of the unilateral cases of H. O. M. gives some very interesting information. Four cases were studied. In the unaffected ear, negative Rinne in no cases. Decreased B. C. in two cases, upper limit decreased 2,000 or more by B. C. in three cases. In all four of the unaffected ears there was some evidence of lesion of nerve or end organ if we include the findings with tuning forks and audiometer.

Conclusions:

I. The determination of the upper limit by B. C. is a far more accurate indication of the condition of the acoustic nerve and its end organ than its determination by A. C.

II. It is usual in acute middle ear infections even in the early stages to find evidences of involvement of nerve or end organ. Rarely with the present methods of diagnosis are we unable to find evidence of such involvement.

III. The lesion of nerve or end organ is not necessarily dependent upon the acute middle ear infection. There is no definite relation between the severity of the middle ear infection and the lesion of nerve or end organ. When the middle ear infection is unilateral the unaffected ear usually presents evidence of a lesion of nerve or end organ.

IV. After the disappearance of the acute middle ear infection there may remain a permanent lesion of nerve or end organ.

V. In every case of chronic otorrhea except one there was evidence of a lesion of nerve or end organ. There is an interdependence between the middle ear lesion and that of the nerve or end organ.

VI. In the H. O. M. cases every ear studied save one presented evidence of lesion of nerve or end organ.

VII. In H. O. M. there is no constant relation between the extent of the lesion in the middle ear and the lesion of nerve or end organ.

THE ESTIMATION OF THE MINIMUM AUDIBILITY OF TONES.

By J. GORDON WILSON, M. D., Chicago, Ill.

The observations made within recent years by physicists on the minimum audibility of tones have awakened an increasing amount of interest among otologists who have been quick to recognize the important bearing such knowledge will have on their specialty. In introducing this subject it will be advantageous in a few words to define the scope of the problem as it presents itself in this discussion.

THE PROBLEM

It is scarcely necessary to say that this discussion is not concerned with the psycho-physical problem of how distinctive qualities of tones are aroused. As we are dealing so far as possible with pure tones, the resultant vibrations from several tones such as are transmitted from an orchestra as one complex (seen for example in phonographic records) and their analyses in the internal ear, do not concern us. Again, inasmuch as the human voice and speech though dependent on a pronounced fundamental are efficiently modified by distinct overtones present, the minimum audibility of the voice will not be referred to. The topic before us represents the basic problem and it is of far reaching importance in otology. On it later the other problems may be advantageously based. We recognize that the quantity of the impulse over a nerve may be increased or diminished; we are here dealing with the minimum quantity required to awaken recognition of a pure tone—that is the threshold of hearing. This threshold may be lowered by education, as is touch in the blind; but I am not concerned with this question but with the average minimum perception in the average individual.

It has long been recognized by physicists that the estimation of the minimum audibility of tones (the threshold of hearing) at all pitches is of prime importance to an understanding of the physics of audition. Within recent years the physicist has encountered many practical problems to which he has sought to apply such knowledge. The late war in this realm as in other realms of physics stimulated the energy of the pure scientist to practical problems in acoustics. We all recall the immense amount of work done to perfect apparatus to detect the presence of and locate the position of submarines. Again, the telephone service, now become one of the necessities of modern life, needs for the engineering of an adequate service a knowledge in absolute terms of the sensitiveness of the hearing of the average telephone user, and this has required in the telephone service a study of the threshold of tone perception.

Not only is this estimation important for the physicist but in otology the testing of the absolute sensitivity of the ear at various pitches is of importance: (1) To determine the degree of impairment of hearing; (2) to localize the pathologic changes, in which it has proved superior to our ordinary methods of testing, and (3) to estimate the results of our treatment.

The difficulties encountered in testing hearing are considerable, much greater than in testing sight. In testing sight we can use one standard, white light. In testing hearing we have no uniform sound to use as a standard. While it is true that both light and sound are vibrations, the eye is not able and is not called on to resolve light into its composite colors, and even if it were the range of such vibrations is small compared with the ear. In the ear such differentiation of vibrations is an inherent part of its function as an organ of sense.

For an accurate knowledge of the degree of hearing a patient possesses we must have an estimate of the range of hearing (quality of hearing) and the quantitative degree

at each pitch. It is known to all of us that hearing for one part of the scale may be seriously impaired while that of another part is hardly affected. Accurate results therefore can only be obtained by estimating quantitatively the hearing for simple tones at all pitches. When one remembers that the qualitative range may be estimated at from 20 cycles to 20,000 cycles, or higher, one recognizes the wide area to be covered. The difficulty of obtaining a suitable apparatus capable of examining the threshold of hearing over so great a range has proved the great barrier to this investigation. This barrier has now to a large extent been surmounted and we are entering on a new era of advance and only a rash man will predict what the final outcome will be.

In such an investigation there are three main questions or problems which present themselves and which may thus be outlined briefly:

(1) What is the minimum amount of energy required to produce the perception of pure tones at various pitches—the physical problem.

(2) What light do such determinations throw on the ear as an organ of hearing—the physiological problem.

(3) Given a mean audible threshold what variations take place in defects of hearing, this involving the further question at which pitches such variations occur,—otological problem.

In considering these questions it is obvious that the more perfect the instrument from the standpoint of the physicist the more accurate the knowledge obtained in regard to the minimum audibility. To obtain the threshold value in the normal ear it is obvious that a prerequisite is that the instrument used be capable of reducing the pitch over the scale beneath normal audibility. There are many factors which will vitiate the findings, e. g., resonances in the telephone receiver, the presence of extraneous noises tending to confuse the observer, either from friction in the machine or in the surrounding media, etc. To recognize and to

eliminate such factors has not been without difficulty. While one cannot say that all of these have now been got rid of, yet I feel assured that in the instrument I know best, the audion oscillator, we have been able to reduce to a minimum such factors as will greatly vitiate the results. Sufficient work has been done by independent observers which correlated with our own, enable one to present results with some assurance. With the machine at our disposal we have been able to approximate so closely to a measurement of the threshold that one can show curves which in the main outline the sensitivity of the ear at the pitches tested. It appears unnecessary to wait till the perfect machine arrives. Such a machine may be necessary for perfect physical measurement. Even with an instrument less perfect than ours, a mean can be obtained from the examination of several normal ears and the variations from the mean obtained by the same instrument under the same external conditions will be approximately correct for all ears so tested. Of course the more accurate the instrument the more accurate the information obtained.

HISTORICAL PART

In the past the physicist has largely worked in his special field undisturbed by any criticism or suggestion the otologist might offer. The otologist has not hesitated to acknowledge his great indebtedness to the physicist. It was the great physicist and physiologist, Helmholtz, who gave us the classical work on hearing. In 1862, Helmholtz published his "Sensations of Tone" and propounded his resonant theory of hearing—a theory modified by himself in later editions as new anatomic data were added, a theory which in spite of many objections still influences the work of physiologists and otologists, and in which the central idea of analysis of sound by resonance has the support of most of them. From the stimulus of that work most of our great aurists of the past drew inspiration, and on its results they explained many of their clinical findings. Helmholtz readily acknowledged and readily used in his

work the findings of the anatomist and naturalist. The problems he studied did not seem to require co-operation with an otologist. It is interesting here to note that the work now to be described has added one other and new objection to the resonant theory. This objection I shall briefly state in the words of my collaborator. "It was possible in the case of this patient to apply in the frequency region of 3,300 d. v. an energy 10 billion times as much as corresponds to normal hearing at this frequency without stimulating in the slightest degree the sensation of hearing in the region below 1,200 d. v. where the patient's hearing was practically normal. Now in the Helmholtz theory, pitch perception is due to the coincidence of the impressed frequency with the natural frequency of the fibers of varying length and tension which constitute the basilar membrane. It is, however, extremely difficult to see how a mechanical system such as that represented by the basilar membrane with its fibers and attached Corti's arches could possibly receive ten billion times the normal energy at a given frequency like 3,300 d. v. without imparting as much as one billionth of that energy to the adjacent fibers which corresponds to vibration frequencies below 1,200 d. v. If these latter fibers had in this case been thus forced into response with one billionth or even one hundred billionth of the incident energy, the patient would have had the sensation of hearing in the region below 1,200 d. v. If these considerations are fatal to the Helmholtz theory, there appears to be no alternative at present but to make pitch perception a property of the nerves themselves, including their endings, without attempting to find a mechanical explanation. This amounts to nothing more than the assertion of our inability at present to obtain a satisfactory mechanical basis for pitch perception." John P. Minton, Ph. D., *Physical Review*, Vol. 19, No. 2, Feb. 1922, p. 95.

Though Helmholtz was not specially interested in the problem of the threshold of hearing, this phase of the subject attracted the interest of several of his successors. Very important work along this line was done by Lord Rayleigh.

To determine the threshold of hearing, for high notes he used a whistle mounted on a Wolfe bottle attached to which was a siphon manometer to regulate a constant air pressure. Having ascertained the distance at which a tone could be heard under as far as possible constant atmospheric conditions, and having calculated the energy emitted by the whistle from the pressure in blowing it, he was able to approximate to the amplitude required for the hearing of the tone. One serious objection was the distance the observer had to go from the source of the sound—in the case quoted 820 meters—involving the disturbing influence of atmospheric factors. To estimate the minimum audibility of low notes, Rayleigh used tuning forks which enabled him to approximate the sound to the ear. From the difference in the decay constants of the fork it was possible to calculate approximately the energy emitted. Rayleigh also used a telephone receiver as the source of sound. "The deflection of the diaphragm for a direct current was measured microscopically and this was considered the same as for alternating currents flowing through a receiver, currents having a period far below the natural period of the diaphragm. With the volume of air in the meatus known it was possible to calculate approximately the change in pressure in the ear drum from the current flowing through the receiver." In reading over Lord Rayleigh's work one readily appreciates the great advantage the audion oscillator offers for such an investigation and learns once more that advances in science depend to a large extent on improvement of instruments of attack.

The German physicist, Wien, investigated this problem by a different method and in 1889 published his results. He made use of the siren to estimate the minimum audibility of sound. The siren, one of the oldest instruments to produce a continuous series of sounds at different pitches, has holes at equidistance or teeth at equidistance. In the former, air is drawn through to produce sounds varying in pitch with the rapidity of vibration of perforated drums; in the latter, the electric siren, the make and break of a

current produces the tone varying in pitch with the rapidity of the make and break. Wien used the electric siren conveying the sound to the ear by means of a telephone. By assuming that the amplitude increases proportionately with the current he calculated the amplitude of the vibration for a tone at the threshold of audibility. His apparatus enabled him to observe results through a range of frequency from 50 to 15,000 cycles. As a result he placed the maximum sensitivity of the ear to sound at about 1,200 to 2,000 d. v., from which area the sensitivity dropped off rapidly on both sides. These results, extensively quoted by physiologists and otologists, especially in Germany, do not appear to be generally accepted by physicists. Two objections urged against the findings of Wien it is well for us now to keep in mind: (1) that the type of telephone receiver used introduces resonances affecting the findings of sensitivity in certain areas; (2) that the siren is not a suitable instrument to determine minimum audibility since the friction necessarily produced by its rotation introduces too many accessory sounds and further in the note of the siren overtones are prominent.

From this time on many physicists attacked the problem either by similar methods, eliminating objectional factors, or by other means; for instance, Kranz of the Sabine Laboratory used a thermal receiver as a source of sound. What concerns us more particularly is that only within the last few years do we find a systematic endeavor to get a closer co-operation of otologist and physicist. Even now many of the results published by physicists are based on the assumption that an ear which to the physicist appears to hear normally is therefore a normal ear, a dictum with which few of us will agree.

In the past there have been otologists with sufficient knowledge of the science of acoustics to appreciate the importance of the problems involved and with skill sufficient to attempt to express this threshold in physical measurements. For instance, Gradenigo attached to the upper end of a fork a dark triangle on a white background. When the fork is struck there is a double vision. One sees two pale triangles with a black tri-

angle common to both; the greater the amplitude the greater the space separating the two triangles; as the amplitude diminishes the two triangles merge more and more into the single black triangle. By noticing the extent of the field when the patient ceases to hear (marked by a millimeter scale), Gradenigo estimated the ratio of hearing. This method, however, is only applicable to low notes.

COOPERATION OF OTOLOGIST AND PHYSICIST NECESSARY.

My object in thus briefly sketching the work of the physicist has this for excuse, that I believe that at present and for some time to come the ascertaining of the threshold of hearing must be done primarily by a physicist but will have its best fruit when done in conjunction with an otologist. Few otologists have the necessary physical and mathematical training to understand the physical problems involved and to calculate the energies necessary to get correct results in estimating the minimum audibility of pure tones either in normal or defective ears, and this to my mind is at present the essential problem. Few of us have any conception of the infinitesimal amount of energy required for the ear to hear. Mr. Minton has calculated that the energy consumed by a 40 Watt lamp would operate a string of telephone receivers which if placed a foot apart, would encircle the globe 25,000 times and yet produce a tone of 1000 cycles that could be heard in each receiver if placed to an ear. The intensity of the sound transmitted to the ear is proportional to the energy. If the ear were a perfect physical instrument this would hold for the amount of energy transmitted to the nerve ending. But there does not seem to be the same relation between the amount of energy transmitted to the ear and that transmitted to the nerve endings; certainly in defective hearing this ratio is greatly altered at different pitches and to a varying degree.

DEFECTS IN OUR PRESENT METHODS AND KNOWLEDGE.

In spite of the large amount of work that has been done especially during the last 25 years to determine in absolute terms the minimum amount of sound, the amplitude of vibration that the ear can perceive, the resulting figures have been most dis-

crepant. The results of the various observers show a wide range of difference. One cause contributing to this was the inadequate apparatus available; another that no otological tests were made to ascertain if the ears so tested were normal; to these may be added that normal ears vary very considerably not only with the age of the patient but among individuals of approximately similar ages, and even between the ears of the same individual.

At present we depend almost entirely on tuning forks. The overtones in the lower forks are got rid of by clamps and in the higher forks are so much higher that they do not interfere with our present application of the test; we are also aided by the fact that the fundamental tone persists longer than the overtones. Tuning forks, however, have great limitations. They are wanting in scientific accuracy because we cannot well measure the amplitude of their vibration. Each vibration period varies in each set and alters somewhat with use. It has been impossible to give a percentage estimate of the impairment of hearing in one ear compared with normal by means of forks, for the amplitude of the tuning fork when it ceases to be heard in the two ears is difficult to estimate.

The otologist has at present no satisfactory means of measuring the intensity of the sound perceived. There is no certainty in regard to the position in the sound scale in which the maximum sensitivity for sound perception lies.

Our present tests with forks, with whistles, etc., give us a rough and ready method of ascertaining fairly accurately whether the lesion lies in the conducting mechanism of the middle ear or in the nerve including its endings. Such a wide generalization has served in the past, but leaves much that is important unanswered. There are few of us fully satisfied with the results obtained and most of us recognize the great gaps in the knowledge of the lesion. Our tests frequently fail us when we desire definitely to locate a lesion. Consider for a moment lesions in the conducting mechanism. Supposing the drum membrane be indrawn and have its curvative altered, we are not able to say how such an alteration will affect the hearing, whether the deficiency in hearing present be

due to this or to some added deficiency of movement of one or more ossicles. If the membrane be relaxed we are unable to state how much deficiency of hearing hereby results or the pitches involved. Such questions present themselves as what defaults in hearing are associated with perforations in various quadrants of the drum—quantitatively and qualitatively; in combinations of middle ear and nerve deafness, which factor predominates and to what degree. It has been stated that in the loosening of the malleus-incus point not only are the low notes diminished but also high notes of plus magnitude; the question arises what is the picture of ankylosis of these joints. But one need not stress this phase of the subject.

METHOD OF TESTING WITH THE AUDION OSCILLATOR.

This method of testing the sensitivity of the ear or the degree of deafness consists, in its essential features, of measuring the minimum audibility current at various frequencies through the vibrations of the diaphragm of a telephone receiver adapted for this purpose. The intensity of the minimum audible sound acting on the ear is proportional to the square of this current. These minimum audible current readings are taken over a range of frequencies from 100 to 4,000 or 5,000 cycles, and the results plotted in the form of curves. Observations have been restricted chiefly to these ranges for two reasons: (1) They include the pitches most important for voice perception and (2) the telephone receiver specially constructed for this work was timed to a natural frequency of over 5,000 and so errors from this source were less likely to vitiate the findings.

The Audion Oscillator is used to supply the current to the telephone receiver, which is placed against the patient's ear at a constant pressure. The oscillator is set for the desired frequency and the current flows through the bridge circuit composed of non-inductive resistances, *a*, *b*, *c*, *d*. The resistances, *c* and *d*, are generally held constant while *a* and *b* are adjusted so that there is sufficient current through the receiver for the patient to hear without effort.

A short circuiting key when depressed, short circuits the receiver and when the patient wishes to listen he releases the key, listens very closely for a second or two and then depresses the key. The patient nods his head if he hears the tone in the receiver. To reduce the tone in the receiver the operator adjusts the resistances *a* and *b* and a slide wire resistance, so decreasing the current through the receiver. The patient again listens as before and indicates if he hears. In this way the current through the receiver can be continuously decreased until the minimum current to make the tone just audible to the patient is obtained: about the vanishing point of the tone the patient is required to listen intently for two or three seconds several times with two or three second intervals between each listening period in order to avoid fatigue. Having obtained the settings of the resistances for minimum audibility at various frequencies the current through the receiver is calculated by means of well known formulae from the values of the resistance of the bridge arms, the impedance of the telephone receiver and the total current indicated by a galvanometer. With these current readings and the vibrational characteristics of the special receiver, the vibrational energy at the various frequencies of the receiver diaphragm can be calculated. Taking the sound intensity as proportional to the values of the vibrational energy we can plot a curve showing the sensitivity of the ear at various frequencies where the sensitivity is considered to be equivalent to the reciprocal of the vibrational energy of the receiver diaphragm for minimum audibility. N. B.—A fuller description of the audion oscillator is being prepared and will be published separately.

To secure the exclusion of outside noises a sound-proof booth has been built in which the patient is seated during the testing. The booth is always used for testing normal ears and for testing ears which depart little from normal.

METHOD OF PROCEDURE

Previous to examination with the audion oscillator the

history of the patient is taken and details noted of previous trouble in the ear, nose and pharynx. The pharynx, naso-pharynx and nose are examined and the permeability of the tubes noted. The drum membranes are carefully observed and a record made of the Rinne and of the Weber tests and of bone conduction. The distance at which the whispered voice is heard is ascertained and fork tests are made to ascertain their diminution or increase from normal. In addition, in ears with defective hearing an examination of the tubes is made with the catheter and a careful examination of the naso-pharynx with mirror or naso-pharyngoscope. When indicated, X Ray plates have been made.

LANTERN SLIDES TO BE SHOWN

In ears with normal hearing the curves are shown plotted in two ways: (1) In terms of relative receiver currents—relative to a mean of several normal ears. (2) In terms of the vibrational energy of the diaphragm.

In ears with defective hearing the curves are plotted relative to an average of several normal ears in terms of relative receiver currents.

SUMMARY OF RESULTS

I. MINIMUM AUDIBILITY IN NORMAL EARS

The variety and irregularity of our results in ears with normal hearing were unexpected. You will have noted that while there exists a considerable variation among normal ears, each ear possesses one or more well defined peaks. The curves show these peaks of maximum sensitivity at various pitches but chiefly near 1,000 cycles. It will be noted that an ear may and usually does possess more than one well defined peak but that when two or more peaks are prominent it usually happens that one markedly overtops the other. While the maxima of sensitivity show great variety in pitch position in different individuals were one to indicate the position in the pitch scale where the maximum sensitivity lies in the average individual, one

would not go far astray if it were placed about 900 cycles.

It does not seem advisable to enter into details on this subject at this time; a complete report is being prepared and will be published later. It is to be understood that this subject is yet in the experimental stage and no deductions are offered. We do not know why certain ears have their maximum acuity as low as 500 or 600 cycles—others as high as 1,500 or 1,700 cycles. Age probably is a factor but it does not appear to be the only factor. While our greatest sensitivity was in a young man of 22, who at 900 cycles had a sensitivity of $1/750 \times 10^{10}$ ergs (an erg being the unit of mechanical energy), yet we found in a man of 35 years at 700 cycles had a sensitivity of $1/220 \times 10^{10}$ ergs. One interesting question for the otologist of the future will be the after history of ears with this extreme sensitivity.

II. MINIMUM AUDIBILITY IN EARS WITH DEFECTIVE HEARING.

In the immediate future it is the study* of the curve of the abnormal ear that offers the most fruitful field and it is in this field that we as practical otologists are most interested. Glimpses of these possibilities have already been given in the papers of Dr. Dean and his collaborators and in the papers of Mr. Minton and myself. With the modified siren used by Dr. Dean and Mr. Budge, as well as with the audion oscillator used by Mr. Minton and myself, a mean audibility can be readily obtained in ears with normal hearing. In the results here shown a base line is drawn to indicate this mean and the values of the relative receiver current necessary to get the threshold at various pitches plotted relative to this, give a curve indicating the defects in hearing. A direct comparison of the sound intensity necessary for hearing in the abnormal ear compared with the normal can be got from the square of the relative receiver current, as the sound intensity is proportional to the vibrational energy of the diaphragm.

At present the curves from the Iowa laboratory and those from the Chicago laboratory are dissimilar. In considering

this question it must be remembered that a different method of plotting the curves has been used. In the papers issued from the Iowa laboratory the curves are plotted in numbers designated in terms of electrical resistance in the apparatus used and the ordinates are not proportional to the current. Our papers show the curves plotted in terms of relative receiver currents. The results should not vary greatly according to the machine used, though presumably they will vary somewhat according to the result obtained in normal hearing tests. But it is well to have at this stage a variety of testing apparatus; one day we will be able to decide which one is best suited for the purpose.

From our examination of curves obtained from ears with defective hearing we present no deductions. When we have sufficient data we shall begin to synthesise. Even now, however, the graphic pictures of some of these curves speak for themselves, for instance, the curves of nerve deafness. The complication of other curves as in otosclerosis make us hesitate to commit ourselves to any deductions.

The observations which I presented to you at our last meeting in regard to the position of tinnitus relative to nerve deafness have been confirmed by observations during the yast year—some of which have been shown. They throw an important light on the pathogenesis of tinnitus—at least in some ears.

One word in conclusion, we all recognize the amount of pessimism which exists not only among otologists but even among the general laity in regard to the outcome of the treatment of hearing defects. Such pessimism is not without cause. Yet this National Otological Society must be heartened by the character of this work being done not in one city but in various centres on this continent. The close co-operation between physicist and otologist is producing lasting results—results which can only be appreciated by one who has taken part in this co-operation. Such co-operation has come to stay and ought to be available to each of us. With the accumulated knowledge which will come, the cloud of pessimism which now oppresses us will be at least partly dissipated.

SUMMARY

I. An accurate means of ascertaining the minimum audibility of tone perception in ears with normal and defective hearing is needed in otology.

II. The results of testing by means of the audion oscillator surpass the means at present at our disposal.

III. By it a permanent record can be made of the amount of hearing present, of the progress of the disease and of the results of treatment.

IV. The method is full of promise for a more accurate diagnosis of the localization of lesions. It gives an explanation of tinnitus. This method offers a new route to investigate the physiology of hearing.



MAY 18 1958

HB

WV 5 A512p 1922

51310760R



NLM 05267745 0

NATIONAL LIBRARY OF MEDICINE